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OF FRUIT
DISEASES

Hesler and Whetzel

THE RURAL MANUALS

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MANUAL

OF

FRUIT DISEASES

BY

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PREFACE

It is the common opinion of authorities that fruit-growers lose millions of dollars annually on account of diseases of their crops. It has been estimated that 75 per cent of this loss may be prevented by spraying — the chief method of fruit-disease control.

In order to understand and to perform properly the measures of fruit-disease control, it is essential that some knowledge of the cause of the disease in hand be acquired. In recent years scientists and laymen alike have recognized the importance of certain technical details regarding the cause in order to undertake the prevention of these losses. Similarly, the value of other facts must be apprehended, such as the history of a disease, where it originated, with what rapidity the pathogene has spread, the losses it is capable of incurring, and under what conditions these destructive outbreaks (epiphytotics) occur.

As evidence that the practicing agriculturists are rapidly becoming acquainted with the value of scientific knowledge regarding diseases of plants, it is only necessary to point to their interest and cooperation in the matter of obtaining accurate information under field conditions. The prejudiced and critical attitude of the grower is now for the most part of no consequence. Little self-protection is now needed by the experimental plant pathologist; the grower's attitude is no longer antagonistic, but he is friendly and, what is more encouraging, he seeks with confidence the advice of the phytopathologist.

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At present, circumstances do not permit the plant doctor to call in person or by letter to advise with every fruit-grower regarding his many problems. No experiment station can furnish bulletins that will answer the purpose in every detail. And up to the present there have been no American texts or reference books dealing wholly, and in some detail, with the diseases of fruits. It is hoped that this Manual will give to the fruit-grower such information on fruit diseases as is available to date. The authors realize, however, that the best possible book on fruit diseases cannot entirely meet the situation. Possibly the day will come when each neighborhood will be provided with its own plant doctor; until this time the advice must remain qualified.

It may appeal to the critic that many technical details are included which will only confuse the grower. But the time has come when the grower and the plant pathologist must meet on an equal footing. No longer should the grower refuse to understand the philosophy of his operations. Any careful consideration of a disease involves the use of certain terms not common in the farmer's vocabulary. The terminology of the plant pathologist should be no more bewildering than that of the family physician or the veterinarian. To assist in an understanding of the usage of certain terms in the text, a glossary is appended. These terms may be found in dictionaries, but differences in the shades of meaning often lead to confusion.

The grower is in the habit of grouping his troubles as well as his labor and projects. In his own mind he classifies diseases on the crop basis. Accordingly, only fruits are included in this Manual. It is hoped that other crops may be the subjects of similar manuals in the future. In seeking guidance on any disease-control problem, the fruit-grower groups fruit diseases according to the fruit. It has therefore seemed best to discuss the diseases of a given fruit in one place. The arrangement of the fruits in the text, that is, alphabet-

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ically, is simple and therefore useful. The authors have attempted to discuss the diseases of each fruit somewhat in the order of their general importance and occurrence in the United States. Obviously, however, this arrangement is difficult to follow.

Some difficulty has been encountered in compiling certain parts of this book. The authors have had no opportunity to become acquainted with several of the diseases under field conditions. But in some instances inaccuracies have been eliminated by a careful criticism of the manuscript by competent men from other sections of the country. Many other parts of the manuscript have been read by colleagues in the Department of Plant Pathology at Cornell University. To the following contemporaries the authors would express their appreciation for valuable assistance along this line: Dr. H. M. Fitzpatrick, Mr. E. F. Hopkins, Professor H. S. Jackson, Dr. W. H. Rankin, Dr. Donald Reddick, Dr. J. R. Schramm, Dr. V. B. Stewart and Dr. J. L. Weimer. For specimens from which some of the photographs were made, and for other accommodations, the authors would make acknowledgment to the following: Dr. J. T. Barrett, Professor H. P. Barss, Dr. Charles Brooks, Mr. H. F. Dietz, Mrs. L. R. Hesler, Dr. F. D. Kern, Professor W. H. Lawrence, Dr. T. F. Manns, Mr. G. W. Martin, Mr. J. W. Roberts, Professor W. H. Sackett, Mr. F. N. Wallace, Dr. F. A. Wolf and Mr. L. A. Zimm. For photographs loaned to the writers due credit is given in each case.

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CORNELL UNIVERSITY,
ITHACA, NEW YORK,
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MANUAL OF FRUIT DISEASES

CHAPTER I

APPLE DISEASES

APPLES, wherever they grow and of whatever variety, are subject to diseases and injuries of one kind or another. Very few varieties are famous because of their marked resistance to all, or even one, important disease. Growers are fully aware that many varieties, both standard and fancy, are by no means scab-proof. In the warmer states, the Yellow Newtown and Ben Davis are highly susceptible to bitter-rot. Stippen, or bitter-pit, one of the most important of apple diseases, is most common on the Baldwin and others of the best varieties of apples. In the northeastern United States, the Twenty Ounce has come to be known as a canker variety; it is invariably affected when other neighboring varieties stand free from this type of trouble. Likewise the Tompkins King, Grimes and others suffer unusually from collar-rot. But some varieties are markedly less sensitive to certain troubles than others. The York Imperial and Grimes are relatively resistant to bitter-rot, and many other prominent examples might be cited.

There are no less than a dozen very important diseases and injuries to which apple-trees are subject, any one of which may bring about considerable annual loss. Many minor diseases,

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often overlooked, bring the total dollar loss far above the fancy of fruit-growers. It would be difficult to set a figure which would represent, accurately or even approximately, the losses incurred each year. A few well-known examples may be tabulated in order that some notion of the gravity of this source of waste may be gained. One authority estimates from reliable figures that there is an average annual loss of more than $40,000,000 in the United States due to the failure to spray apples. Such devastation, of course, is brought about both by diseases, chiefly caused by fungi, and by insects, and it would be quite impossible to assign the losses to the proper offender in every case. Individual examples of losses follow: (1) bitter-rot wrought damage to the apple-crop of the United States in 1900 amounting to $10,000,000; (2) black-rot canker induces an annual loss of $750,000 in New York; (3) blotch is said to have caused in one county in Arkansas, in 1906, a loss of $950,000; (4) rust, in 1912, was so destructive in West Virginia that actual fruit-losses ranged from $2000 to $3000 an orchard.

Apple diseases are important because of the nature of the injury inflicted. Losses do not stop with damage to, or destruction of, the fruit. Blossoms may be killed; the set of fruit may be dropped; woody parts including twigs, limbs, trunks and roots may be blighted, cankered or rotted; leaves may be spotted and even dropped prematurely. It is indeed fortunate that most of these types of troubles may be reduced to a point where fruit-growing may still be carried on with pleasure and profit.

Most apple diseases are caused by fungi, but a few result from the detrimental action of bacteria, and certain environmental factors. The best-known, the most cosmopolitan, and the most important of apple troubles generally is without question the fungous disease known as apple-scab. Other diseases and injuries affecting this fruit are discussed somewhat in order of their general prevalence and importance.

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Scab

Caused by *Venturia inaequalis* (Cooke) Winter

The scab disease of the apple is universally the best known of all fungous troubles affecting this fruit. While it attacks only the apple and certain closely related species, its ubiquitous nature accounts for its prominent rank among the diseases of its kind. This familiar trouble is commonly thought to be the same as pear-scab, but these two diseases, while similar in their symptomatic and causal aspects, are not identical. Nor should the scab diseases of the peach, cherry, and citrus be confused with the scab of apple.

All varieties of apples are affected, some more severely than others. A variety may be resistant in one year and susceptible in another, under conditions which in both cases are apparently favorable for scab on the average susceptible variety. For example, the Baldwin is usually listed among the resistant sorts; yet in 1910 this variety showed in certain localities 98 per cent scab on unsprayed trees. The Ben Davis, also said to be resistant, often shows as much scab as the average variety. A suggested explanation of these conflicting observations is found in such factors as the color of the fruit, the relation of weather conditions to the development of the fruit, and the adaptation of the pathogene to new conditions. Scab lesions are not so conspicuous on dark-colored fruit as on lighter varieties, and thus the Baldwin bears the reputation of being resistant, while the Rhode Island, which perhaps is really the more resistant of the two, is regarded as very susceptible. Further, in connection with the weather, in a given year the Baldwin may be at its most susceptible stage at the time when weather favorable to infection prevails; that year the Baldwin would appear susceptible while in another year it escapes the disease and is then classed as resistant. This suggestion may

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well apply to other varieties. And finally, the evolution of the pathogene often keeps pace with that of the host. With the development of resistant or immune strains or varieties come eventually strains of the pathogene capable of attacking them.

While this disease is generally known to American growers as scab or apple-scab, it is frequently called the fungus. In Australia and South Africa it is spoken of as black-spot. The disease is unquestionably of foreign origin and probably has been peculiar to the apple since that fruit was brought under cultivation. The first records came from Europe in 1819. It was subsequently reported from France in 1829, having since attracted attention in nearly all countries where apples are grown. The disease was first recorded in America from Pennsylvania and New York in 1834. For a time apple-scab was reported to be absent in the apple-growing valleys of the Pacific Coast and Rocky Mountain states, but within the last ten years it has in those regions become an important factor in apple-culture. While now having a general geographical range over the United States wherever the apple is grown, it is most destructive in the cooler regions of eastern and northern United States, the northern Mississippi Valley, the northern Pacific Coast regions, in the apple-sections of Idaho and Montana, and in the mountainous regions of Virginia, Arkansas, and certain other southern states.

Apple-scab may be said to be the most important disease of this fruit in northern United States and in southern Canada. In the Mississippi Valley, north to central Illinois, Indiana and Ohio, other diseases, especially bitter-rot and apple-blotch, are close competitors, and in many seasons are far more important than scab. It has been estimated that the average annual loss in New York State due to failure to spray the apple-crop is not less than three millions of dollars, and for the United States there is a corresponding loss of over forty millions. Not infrequently there is a total loss from failure of fruit to set due to this disease.

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The losses are greatest in epiphytotic years, but these occur with sufficient frequency to make apple-growing unprofitable unless preventive measures are taken. The nature of the losses may be indicated as follows: (1) Reduction in or destruction of the set of fruit. Heavy losses of fruit, in some cases total, are incurred under conditions favorable to scab at blossoming-time. (2) Impairing the efficiency of the foliage. Affected leaves are often smaller than normal ones and they may fall prematurely. (3) Reduction in size of the fruit. Scabby apples are almost always smaller than healthy ones. (4) Reduction in quality of the fruit. This is usually regarded as the chief consideration, but obviously other types of losses are nearly of equal importance. (5) The keeping qualities of the fruit are diminished. Pink-rot and other storage troubles commonly follow scab on stored apples. (6) The number of windfalls is increased just before picking time. Scabby fruit does not cling well to the trees.

Symptoms.

The disease affects the leaves, flowers and fruit. On certain varieties, such as the Lady, scab is found on the twigs; but this form of the disease is rare in America.

On the leaves the disease usually makes its first appearance in the spring on the lower surface, since that side is first exposed as the leaf emerges from the bud. These hypophyllous spots are as a rule smaller and less prominent than those on the upper surface. The former lesions are brownish or olivaceous, of a webby appearance, with margins indefinite, fimbriate, and commonly tending to follow the veins of the leaf (Fig. 1). The spots on the upper surface (Fig. 1) are similar to those below, but are more definite in outline, darker, more velvety and larger. When a great number of lesions occur on a leaf, each spot is smaller than where fewer lesions prevail. The spots on the upper surface often cause the affected area to become convex above and concave below. Near the end of the season the

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spots may fuse and frequently the leaf is killed. In case of severe infection defoliation may result. On the blossoms the lesions are found chiefly on the pedicels and calyx. The olivaceous spots may encircle the slender stalk as a result of which it withers and ultimately falls, thus reducing the set of fruit. Blossoms which show even a single lesion seldom hang to the tree. The petals are never affected. Mature scab spots on the fruit are very similar in size, shape and color to those on the upper leaf-surface, although at first they are smaller and more sharply defined. They are usually most numerous about the blossom-end (Figs. 2 and 3) of the fruit, due to the fact that infection occurs largely in the spring while the young apples still remain in an upright position (Fig. 4). The lesions on the fruit show a whitish, papery margin, often very wide; this is the undissolved cuticle which has been slightly uplifted by the pathogene advancing at the edge of the lesion (Fig. 2). As the spots grow

FIG. 1. — Apple-scab on leaves; types of infection on upper (left) and lower (right) surfaces.

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older the delicate fungal coating becomes extinct at the center, and exposed at this point is the brown corky layer of the host-tissue. This frequently becomes checked and cracked. In some cases the whole spot thus becomes corky so that the pathogene disappears entirely, leaving a russeted scar on the side of the fruit. Scab spots often cause a dwarfing of the side of the apple on which they occur. When the attack is severe, the fruit shrivels and falls. The removal of the cuticle permits the entrance of rot-producing pathogenes, and consequently decay of the apple-flesh appears beneath scab spots. Late infections, which appear at picking time, are much smaller, being scarcely more than mere specks; they are also very black and no marked depression nor dwarfing of the fruit accompanies such infections. The scab spots on twigs vary with the variety affected. In some cases the affected portion becomes somewhat swollen, and the twig as a whole becomes prominently blistered. Severely attacked young twigs may appear blistered over the surface to such an extent that the bark will subsequently peel in flakes. This type of injury is common in Europe, and is called grind or scurf by the Germans. In other cases the shoots are not swollen and do not present a blistered appearance. A third type of symptom on the twigs is found in cases where the scab spots are isolated, pock-like markings.

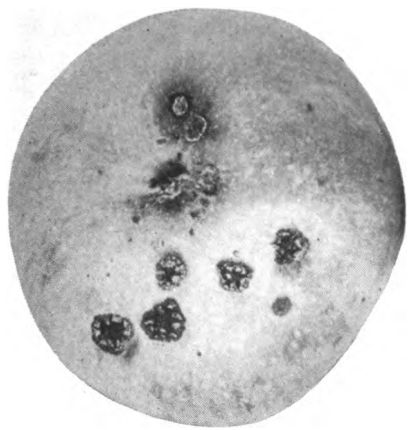

FIG. 2. — Apple-scab, margins of spots showing uplifted cuticle. Lesions about blossom-end.

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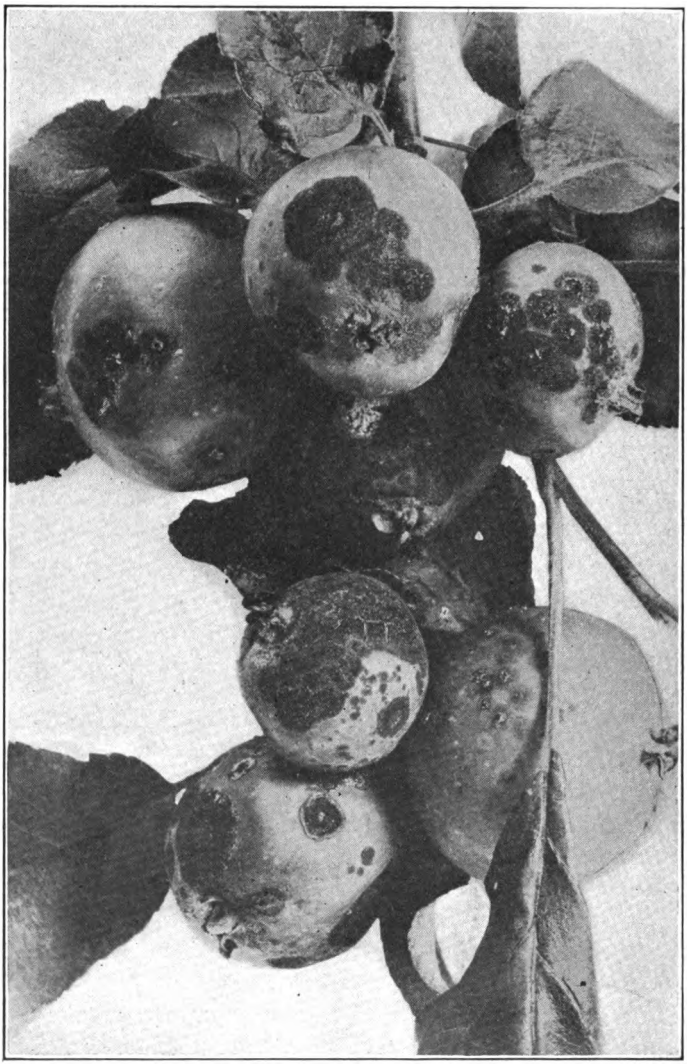

FIG. 3. — Cluster of scabby apples. Many scab-lesions near calyx end.

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Cause of apple-scab.

The apple-scab pathogene is a fungus known by the name *Venturia inæqualis*. It passes the winter in the old fallen leaves as immature perithecia. With the advent of spring these sexual fruiting bodies come to full maturity by the time the blossom-buds are ready to open. These spring activities of the parasite are characterized chiefly by the formation, within the perithecia, of ascospores which constitute the principal inoculum for the first infections. Inoculation is brought about through the agency of the wind. The ascospores are forcibly discharged into the air from the perithecia imbedded in the old leaves on the ground, and these spores, being extremely light, are carried to the opening buds. Spore discharge is conditioned by (1) the maturity of the ascospores themselves. They are maturing during a period of about one month beginning approximately at the time the blossom-buds are ready to open. (2) The occurrence of rain-periods when the ascospores are mature. It has been estimated that in forty-five minutes of wet weather at the proper time no less than eight billion ascospores might be discharged from the old fallen leaves under a large apple-tree. These figures indicate how one may account for even the most abundant primary infection of young leaves and blossoms that has ever been recorded. Since the lower surfaces of the unfolding leaves and the young fruit-pedicels are

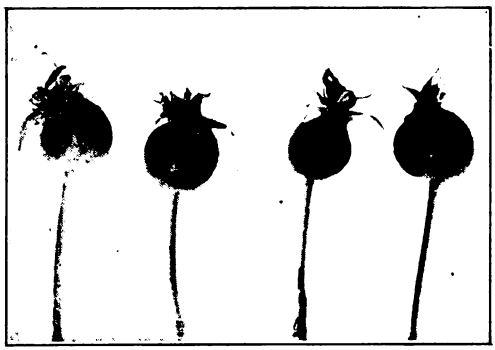

FIG. 4. — Scab on young apples. Lesions on both the fruit and pedicels.

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most exposed at the time of first ascospore-discharge, these constitute the first points of attack. It has been frequently observed that the leaves of the blossom-buds suffer more severely than those of the leaf-buds. This is explained on the grounds that the former buds open first; the leaf-buds, opening later, often escape the early inoculation to which the leaves of the blossom-buds are exposed.

It has been noted that the ascospores are discharged under rainy conditions; and unless this condition prevails for several hours the ascospores cannot germinate, a process preliminary to penetration of the leaves and pedicels. Following a rain any conditions favoring the retention of moisture also favor spore-germination. Several factors acting in this connection are: dense foliage, which prevents prompt evaporation after rains; good air drainage favors this evaporation and accordingly those trees on the hill-tops are less liable to scab-infection than those in low pockets; showers followed by winds are unfavorable to infection since the moisture is quickly removed from the leaves, while showers in the evening followed by a calm night are highly favorable to infection. Spore-germination occurs by the emission of a mycelial thread, called a germtube, which penetrates the leaf to a slight extent, usually not going deeper than the cuticle as long as the leaf hangs on the tree. Sometimes the first layer of leaf-cells, the epidermis, is invaded. Within a very few days the pathogene establishes a food relation with the host, the lesion becoming visible to the naked eye within two weeks or less. The germtube, as a result of extensive growth, becomes a dense system of branching threads — the mycelium. These threads grow radially from the point of invasion, partially dissolving the cuticle as they proceed. The undissolved cuticle at the advancing margin is uplifted and gradually breaks away in flakes, forming a protective covering in the form of a papery rim about the border of the spot. While the pathogene undoubtedly gets most of

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its food from the cuticle, water is drawn from adjacent cells as evidenced by the one-sidedness of affected apples and in the puffing of the leaves under the scab spots.

The mycelium of the pathogene on the leaves and fruits modifies its form slightly by transforming into a stroma, a spore-bearing cushion. From this cushion arise spore-stalks, conidiophores, which cut successively from the free outer end several conidia — asexual summer spores. These conidia mature rapidly and are blown to other leaves and fruits, where they produce scab spots just as described for the ascospores. From these spots, caused by the growth from the conidia, arise again the stromata on which a second crop of conidia are borne. These are likewise capable of further spread of the pathogene. This process repeats itself throughout the growing-season, depending on conditions of moisture. If fruits are inoculated just prior to picking, the spots appear in storage; their appearance has been described under Symptoms. Fruits which fall carry the fungus with them, but the pathogene never develops further. Leaves, on the other hand, furnish the chief hibernating quarters for the fungus. As soon as the foliage falls, the mycelium of the organism penetrates all parts of the leaves. During the autumn and early winter the perithecia are partially formed, these organs lying dormant until spring. At this time growth is resumed and the life-cycle is again initiated. There is considerable evidence that the conidia hibernate on the twigs or in the bud-scales, but the old leaves on the ground are to be regarded as the chief source of the primary inoculum.

Control of apple-scab.

The destruction of fallen leaves would appear to lessen the primary infection. But in actual practice that method alone is not reliable and at best is only to supplement spraying or dusting. Spraying is at present the chief method of control. The fungicide used is lime-sulfur solution, diluted one gallon

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of the concentrated solution (testing 32° Baumé) to forty gallons of water. To this is added arsenate of lead at the rate of two pounds (paste; or one pound, powdered) to each fifty gallons of the diluted lime-sulfur. Aside from its insecticidal value, the arsenate of lead increases the fungicidal value of the lime-sulfur. Applications should be made as follows: (1) Just before the blossoms open, but after the individuals of the cluster have separated (Fig. 125, page 440). The period for effective spraying at this time is from one to three days. This application should hold the set of fruit, if the fruit-pedicels are thoroughly coated. This is the most important application of all in a generally wet season. (2) Spray just after the blossoms fall, beginning when they are two-thirds off (Fig. 126, page 441). This application gives a clean crop of fruit. The period for effective spraying is from two to five days. (3) Ten days to two weeks after the second application, depending on the weather conditions. In a dry spring this application may be omitted. (4) The latter part of July or the first of August. Again the grower is to be guided by weather conditions, although ordinarily this application should not be omitted, as a relatively clean crop may be badly damaged by late infections, if this application is not made. Sprayings in all cases should be done before rain-periods, since the fungicide must be on the susceptible part before the pathogene is. Effective fungicides do not wash off sufficiently to destroy their efficiency. By studying the low barometric areas indicated on the daily weather map the orchardist should be able to predict weather conditions for two or three days in advance. A nozzle giving a fine driving mist should be used. The material should be applied at a pressure of 200 pounds. Spraying against the wind instead of with it saves materials and labor and gives a more effective distribution. Spray from a tower of sufficient height to enable the operator to spray the highest parts. Spray every season, never omitting the first, second, and rarely

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the fourth applications. Bordeaux mixture has been used widely for the control of apple diseases, and particularly for scab. It has been replaced with lime-sulfur spray and a sulfur-lead dust on account of the injury or russeting of fruit which results from the use of bordeaux (Fig. 124, page 434).

It has been shown that dusting with properly powdered materials is fully as effective as spraying. The operation is much more rapid in covering the orchard; an orchard may be dusted in one-fifth to one-tenth the time required for spraying the same. This dusting process not only saves time but an orchard of large acreage may be protected at critical times, a thing not always possible with the slower liquid process. Apply a mixture of ninety parts finely powdered sulfur and ten parts arsenate of lead (powdered), using about one and one-fourth to two and one-half pounds a tree at each application. The smaller dusting machines may be operated by hand, the larger ones by a gasoline engine. The time of application of dust mixtures does not differ from that of the application of sprays.

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Bitter-Rot

Caused by *Glomerella cingulata* (Stoneman) Sp. and von S.

While this disease occurs on a great variety of hosts, it is of most consequence on the apple. On this fruit it is almost invariably called bitter-rot, in spite of the apparent unfitness of the name. The affected flesh is not always bitter. The names anthracnose and ripe-rot have also been applied to this disease, but since the trouble occurs on both green and ripe fruits, the latter term is an objectionable one. The name anthracnose is reserved for another disease of the Pacific Northwest. The disease on the limbs is called bitter-rot canker.

It is difficult to determine the origin of bitter-rot, but America appears to be the only country in which apples suffer from it. It was recorded in North Carolina in 1867, and with the development of apple-culture throughout the central belt of states, the range and destructiveness of this disease have gradually increased. By 1887 bitter-rot had become a serious trouble in many eastern states along the thirty-seventh parallel. It now occurs in all the territory east of the Mississippi River, and west including Kansas, Oklahoma, Texas, Missouri, Arkansas and Louisiana. Within this region bitter-rot is most prevalent in a belt of states on the line of the Ohio River, from Virginia to Oklahoma. Epiphytotics in many of the states in this belt have occurred with great frequency and destructiveness.

Bitter-rot is unquestionably the most ruinous of all apple diseases in certain years; this is particularly true of the section where it is most prevalent. This disease, more than any other apple disease, is one to be feared by apple-growers. Its sudden appearance after great expenditures of time, money and energy have been made in producing a fine crop causes it to be

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especially dreaded. A large harvest may be totally lost in a few days. Bitter-rot has probably done more to discourage apple-growing in many regions than all other fungous and insect pests combined.

The losses resulting from this disease are of two sorts; namely, injury to the fruit, and injury to the limbs. Affected fruits are rendered worthless, so far as their market value is concerned, the decay-process being rapid and complete. The amount of the losses incurred in some years has been so great as to cause many apple-orchardists to abandon the business. Instances are on record where bearing apple-orchards have been leased at $5 an acre for a period of five years, the grower choosing to be guaranteed this small sum rather than venture getting nothing from his trees on account of the bitter-rot disease. It is estimated that in four counties in Illinois the loss due to this disease was $1,500,000 in one season. Single growers sometimes lose 10,000 to 20,000 barrels of apples, while the damage to the apple-crop of the United States in 1900 was estimated at $10,000,000. Bitter-rot cankers are destructive, like other cankers, in that the bark is killed. In many cases limbs are girdled and death of the affected member results.

Not all apple-varieties are affected alike. In Virginia the Yellow Newtown (Albemarle Pippin) is preeminently the greatest sufferer. On the other hand, the Winesap is conspicuous because of its resistance. The Ben Davis is said to be one of the most susceptible varieties in the Middle West; in Virginia, however, it shows a comparatively slight tendency to rot. Along the Atlantic seaboard the York Imperial and the Grimes are regarded as relatively resistant. The Willow and Huntsman are listed as susceptible in Missouri and Illinois.

Symptoms.

Bitter-rot may be expected to show on the fruit at any time from June to October, although July and August are bitter-rot months. The time of the first appearance varies with the geo-

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graphical location, with the age and variety of the fruits concerned, and with the weather. It is the rule that a fruit shows but one or only a few spots (Fig. 5); but in cases of severe infection as many as 1200 separate lesions have been counted. Where only a small number of spots occur these continue to enlarge, merging with each other until the whole fruit is involved. If a great number of lesions begin simultaneously on the same fruit, only a few continue to spread, the greater number remaining as small, brown, raised blisters on the surface.

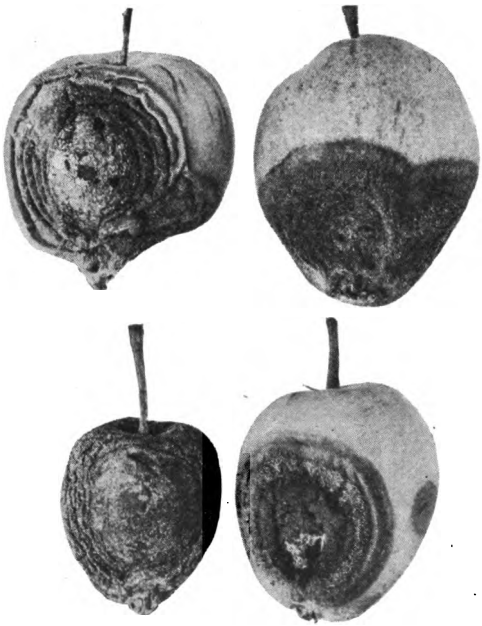

FIG. 5. — Bitter-rot of apples.

A lesion begins as a small light-brown discoloration beneath the skin. It rapidly enlarges and remains firm in texture and circular in form (Fig. 5). The color very soon becomes dark-brown and when about one-eighth of an inch in diameter the rotted area is distinctly sunken and sharply defined. When about one-half of an inch in diameter small black dots appear at more or less irregular intervals beneath the epidermis of the sunken area. These may be arranged concentrically or scattered without order (Fig. 5). Eventually these little black dots, the fruiting pustules of the pathogene, break through the

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epidermis exposing pink masses at their tips (Fig. 5). As the spot increases in diameter the pustules follow rather closely the advancing circumference of the lesion. At the same time the spot is developing outwardly, it is also progressing inwardly, and a section made perpendicular to the surface, through the center of the spot, shows the rotted area to be funnel-shaped. Finally, the whole fruit is involved and a mummy is the result (Fig. 5). Some of the affected fruits fall while others cling to the tree for at least a year. Spots which have been retarded by cool weather have an especially prominent purplish margin. Many late infections are reddish or purplish specks, never developing further on account of adverse conditions.

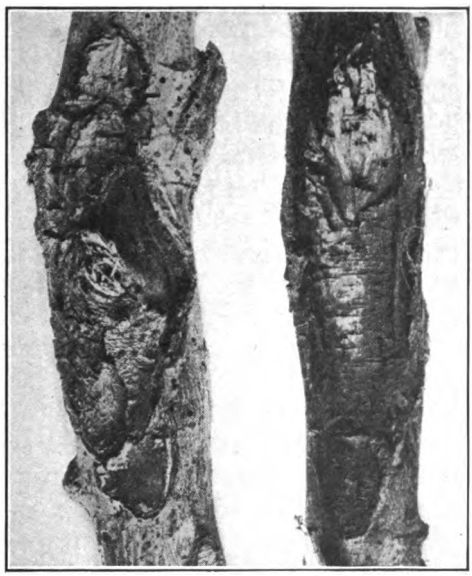

FIG. 6. — Bitter-rot cankers.

Cankers are developed on the twigs, limbs (Fig. 6) and fruit-spurs. Trunks rarely show bitter-rot cankers. Smaller twigs are sometimes wholly and suddenly killed, resulting in a twig-blight. The Jonathan and Willow varieties are more subject to this type of injury than others. Most cankers on the limbs have at their center a dead twig or evidence of one having been there (Fig. 6). The canker is at first a small discolored area in the outer bark, the smooth edges of which are sharply set off,

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oval in shape, measuring one or more inches in length. With increase in size the edges become more or less ragged and the surface is somewhat roughened. The dead bark is cracked and fissured. In some instances it is broken away, although it ordinarily dries out and adheres to the wood. The major portion of the canker is at last clearly depressed, while about the margin of the dead area is formed a callus.

Bitter-rot is not known to occur on the leaves.

Cause of bitter-rot.

The pathogene causing this disease is *Glomerella cingulata.* The fact that the same organism is responsible for both the rot and canker forms of the disease has been fully established. For example, the fungus may readily pass from the bark to the fruit, and this fact is highly important in its life-cycle. Following the seasonal development of the pathogene, it is to be noted that there are two chief sources of the inoculum in the spring; these are the mummies which hang to the tree through the winter, and the cankered areas in the limbs. From these sources conidia are washed by rains to the susceptible parts below, that is, young fruits and limbs. Insects appear to have little to do with the dissemination of the fungus. The best evidence that a canker or a mummy are sources of the inoculum, and that rain is the inoculating agent, is found in the fact that affected fruits usually map a pyramidal area at the apex of which is found a canker or a mummy. Furthermore, infections on fruits are frequently arranged in lines toward the calyx-end, which indicates that the spores were washed by water down the sides of the apple. The conidia soon germinate, their germtubes being capable of entering through the healthy surface as well as through wounds. Abrasions in the bark of various sorts furnish a channel of entrance into larger limbs. The fungus may be able to penetrate uninjured twigs and from these organs it spreads readily into the parent branch forming the canker previously described. Within a few hours after

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inoculation spots an inch in diameter will develop on the fruits; however, the rate of rotting depends on the temperature and the age and variety of the fruit concerned. As previously pointed out, the fungus develops fruiting pustules, acervuli, by the time the spot is an inch or less in diameter. Their general appearance and arrangement has also been described. Within the acervuli conidiophores arise, each stalk being capable of developing several conidia successively at the free outer end. These spores, as they are formed, pile up at the apex of the acervulus and in quantities are pink. These groups of conidia in the concentric or scattered acervuli produce a very striking appearance, which forms one of the most characteristic symptoms of the disease. When moist the conidia cohere in sticky masses for a time, but when wet are easily separated and washed to other points during the rains. Such conidia are in turn capable of inducing bitter-rot and within three to seven days another crop of conidia is ready for dissemination. This process may be frequently repeated from June to October, if weather conditions are favorable. Many of the conidia are carried in rain drops by heavy winds to other apple-trees which thus become new centers of infection. Many conidia, too, are deposited on dead parts of a tree and there germinate, and establish the fungus for the winter. Accordingly it has been shown that the pathogene not only hibernates in the bitter-rot mummies and cankers, but also in the Illinois blister-cankers, dead tips of fruit-spurs, frost-injured portions of limbs, fire-blight cankers, blotch and black-rot cankers. In any or all of these places the fungus overwinters in the form of mycelium. In the spring, the parasite resumes activities by developing acervuli from which the first spring crop of conidia are produced. The fungus has a sexual stage, in which perithecia, asci and ascospores are borne, but it has not been shown that this stage is important in the life-cycle of the organism.

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The history of bitter-rot shows that it has always been spasmodic and erratic in its occurrence in different seasons. But the irregularity is not confined alone to different seasons, for it makes its appearance in different years at different dates. In different orchards it may occur at widely separated intervals during the same season. The fruits on the sunny side of a tree frequently are destroyed first. Sometimes the crop on the south side of a tree is destroyed while on the north side it escapes the disease. Likewise the fruit on the inside, lower branches, being well protected from the sun, often escapes, whereas apples exposed to the sun are ruined. Trees in the shaded mountain hollows of Virginia are said to show less rot than trees in situations more exposed to the sun.

All of these points indicate a decidedly close relationship between the fungus and the weather, especially temperature. The fact that the fungus is confined to the warmer states, that it appears chiefly in July and August, and that fruits exposed to the sun are destroyed first, point unmistakably to the fact that the pathogene is a hot weather fungus. Furthermore cold weather usually checks the disease — or may stop it altogether. A cold spring may retard the development of the fungus at that season, but there usually results a later attack on the fruit. In cool, dry summers bitter-rot is sparingly present. Conditions most favorable for the development of the disease are warm, moist weather. A short series of hot, wet days in August may bring about a sudden and very destructive attack. Nights with a heavy dew, alternating with hot days, are usually followed by an extensive outbreak of the disease, even destroying the whole crop in three days.

Control of bitter-rot.

Cankers, being a chief source of the inoculum, should be removed. Remove the whole limb except where the affected branch is a large and valuable one. Dead parts and cankers other than bitter-rot lesions should be removed. All these

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operations should be performed in the winter since the cankers are not only more easily located when the leaves are off, but such work should be done before the conidia are disseminated in the summer.

Spraying has proved effective, 90 per cent bitter-rot-free fruit being a possibility. Dormant spraying is said to have no value. Spray at least once before the buds open; make a second application by the middle of June. Succeeding applications must be made at intervals governed by the weather. Bordeaux mixture, 4–4–50, should be used; lime-sulfur is not effective.

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Fire-Blight

Caused by *Bacillus amylovorus* (Burr.) Trev.

The apple and the wild crab are commonly attacked by fire-blight. In the nursery these forms are more seriously affected than pears. In the young orchard the disease is also injurious, but old bearing trees do not suffer materially. It is true that the latter class of trees frequently show a large amount of twig-blight, yet the financial loss from such an affectation is negligible. While no variety is wholly immune, there is an apparent

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difference in susceptibility. Under nursery conditions there is evidence that the Yellow Transparent, Golden Russet, Sutton, Fameuse, Wagener, Tompkins King, Rhode Island and others are more affected than Ben Davis, Red Astrachan, Oldenburg and Gravenstein. In New York and neighboring states such varieties of the commercial orchard as Tompkins King, Baldwin, Grimes, Alexander and others are susceptible. In Virginia, the York Imperial and Grimes are most likely to show fire-blight. In this and other regions one of the serious troubles in connection with apple-growing is a form of fire-blight known as collar-blight, collar-rot, or crown-rot. In many cases this type of trouble is a form of winter-injury.

Symptoms.

Large cankers may be developed on the collar of bearing trees, although these do not constitute the first noticeable signs of the disease. The leaves appear yellowish and smaller on one or more of the larger limbs, this effect developing gradually in contrast to the sudden darkening of the foliage of twigs infected with fire-blight. Premature defoliation is induced. The canker is of variable size, depending on its age, and shows a dark, sunken, smooth surface. Cankers in which the pathogene is advancing do not exhibit a distinct margin as is the case with older infections. The lesion develops until midsummer when its growth is arrested, but is renewed the following season. Ultimately complete girdling and death of the tree may result. Usually the canker extends up the body of the tree into the branches; it may also extend down into the lateral roots. In case the collar-rot is due to weather conditions there is no progressive dying as is found in this type of fire-blight. Furthermore winter-injury is first seen at the beginning of the growing season, whereas collar-blight is not observed until midsummer. Fire-blight cankers are known to develop commonly at the base of water-sprouts about wounds of various kinds on limbs and body.

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Cause.

This is a bacterial disease. It is caused by *Bacillus amylovorus* which also attacks the pear, quince, plum, apricot and other trees. The life-history of the parasite on apple may be understood by reading the account under Pear (page 327).

Control.

For trees which are less than half girdled, surgical treatment is profitable. Otherwise, bridge-grafting can be followed as a matter of recourse. A draw-shave, mallet, chisel and farrier's knife are desirable tools for the surgery work. Remove the soil from the affected crown and roots. Cut out all discolored tissue, pointing the upper and lower ends of the wound. Wash the cut surface with corrosive sublimate, and after the wound is dry apply a wound-dressing, preferably coal-tar.

For a full discussion of this disease see Pear (page 323).

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STIPPEN, OR BITTER-PIT

Caused by fluctuating water supply

It is difficult to estimate the importance of this disease. Apples are not destroyed, but their appearance and quality are affected in a manner not easily measured. Figures representing reliable investigation into the amount of losses are not

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available. Not infrequently, however, growers state dogmatically that 50 per cent or more of their crop is affected. Rarely is a crop entirely lost; the fruits are not reduced to absolute worthlessness although in severe cases an apple affected with bitter-pit, or stippen, is extremely objectionable. From the standpoint of losses through reduced quality, the past history of the disease indicates clearly that stippen ranks high among the most important apple diseases of the world. In New York state, at least during certain years, the disease is the most serious of all apple troubles.

Although pears and quinces are known to be affected, yet the apple is by far the most seriously injured. It would not be safe to say that any variety of apple in the United States is immune to bitter-pit. Observations and records show, however, that the Baldwin, above all others, is the most susceptible variety grown in North America. Many growers, in fact, have come to believe that stippen is a disease peculiar to this variety, whence the name Baldwin-spot. But this name is objectionable since the Baldwin is liable to other spot diseases of the fruit. Other varieties prominent in American apple-culture that are commonly affected with stippen are Northern Spy, Rhode Island and Tompkins King. It is a noteworthy fact that the disease under consideration affects most commonly, and often to a grave extent, some of the most valuable commercial varieties, while, as intimated above, a number of other varieties are subject to it.

Like many other diseases of plants, stippen was known to growers long before it was the subject of published writings. There are good grounds for believing that the trouble may have existed in the apple from the time when it was first generally cultivated. In 1869 a pitting of apples was described in German literature. In that country it is now called Stippen, Stippflecke, and Stippigwerden, all of these names referring to a pitting or stippling. In 1886 it was reported from Australia

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and about ten years later the name bitter-pit was given it in that country. In the early nineties it caused alarm among Australian growers because of its obscure and mysterious nature, and finally an extensive investigation was initiated as a result of action on the part of the growers. First authoritative records of stippen in the United States date back to 1891; it was then common on Baldwins in Vermont. Subsequently it was found to occur in Canada, England, France, Russia, South Africa and New Zealand. In fact the geographical range of bitter-pit seems to be coincident with that of the cultivated apple. Most damage is done in America and Australia.

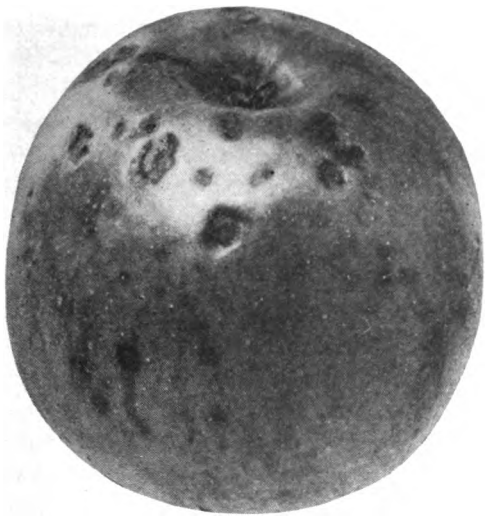

FIG. 7. — Stippen, or bitter-pit.

Symptoms.

The fruit only is affected by stippen. The disease may be found toward the end of the growing-season, at least after the fruit is half grown; or it may not be seen until after the fruit has been placed in storage. Fruits approaching maturity seem to be in the most critical stage of their development so far as bitter-pit is concerned. Its appearance amongst the fruit on a tree is very erratic. Sometimes only one apple on a twig is affected. Again, all the fruits of a cluster are pitted.

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It has, beyond reasonable doubt, been demonstrated by means of the X-rays that the disease develops internally before there are any external signs of it. The first outward symptom of the disease is the appearance of slightly sunken spots here and there over the surface, usually most numerous toward the blossom-end (Fig. 7). These spots or pits tend to a circular form and vary from mere dots to depressions measuring a quarter of an inch or more in diameter. They look like hail or sand bruises (Fig. 7). On a red variety the color is at first darker red, while on a green variety the spots are darker green. Finally the depressed areas are brown. As the fruits mature the pits become more numerous and deeper, but the skin covering them is not broken nor ruptured in any way. Another form of the disease is found in what is known in the western United States as hollow-apple. This form is sometimes called confluent bitter-pit or crinkle because the upper surface of the fruit develops rough folds giving it a crinkled appearance.

If an affected apple is cut open, an area of the flesh directly beneath a pit is found to be dead, brown, dry and spongy, or corky. On account of these characters various names have arisen: dry-rot, apple brown-spot, leige (cork disease), and others. Throughout the pulp of the apple there are usually found brown streaks or spots of dry, spongy flesh which do not extend to the surface of the apple, and which are not connected with the surface lesions. Where the pits and internal brown spots are numerous the apple may have a bitter taste, whence the name bitter-pit.

There are many appearances similar to, or mistaken for, bitter-pit. The apple is subject to several fruit-spot diseases some of which may be regarded as identical with stippen by the casual observer. The New England fruit-spot is distinguished from stippen by its spots being decidedly less sunken, and by the lack of semblance to a bruise. In the fruit-spot one or more black pimples develop on the affected

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area; in the case of stippen there is no evidence of such structures.

Bitter-pit, or stippen, is frequently confused with hail marks, and bruises of various kinds. In the case of any such injuries, however, the skin has the appearance of having been pushed in rather than shrunken, and also mechanical injuries often show a broken skin, a feature not characteristic of stippen. Scab is distinguished as follows: it shows at first as a velvety, then as a corky, lesion, not depressed. The various apple-rot lesions are not depressed until much larger than stippen-spots. San José scale marks are sometimes regarded as bitter-pit lesions, but the former does not show the plain depression of the latter. Most growers will not mistake other diseases for bitter-pit.

Cause of stippen.

Stippen is a non-parasitic disease, one in which the primary cause is not certainly known, but one for which all parasitic organisms, such as bacteria and fungi, are in no way responsible. The common opinion of those who have given some consideration to this phase of the subject is that the pits result from a disturbed water relationship. As to the nature of this disturbance, however, authorities are not entirely agreed.

Since every grower has his own opinion in regard to the cause of this disease it may be of interest to give some of the theories of those who have most carefully investigated it. In order to make these theories clear it is first necessary to have clearly in mind the general structure of the apple-fruit and the nature of the spots. The flesh of the growing apple is made up of rather large egg-shaped cells within which the living substance of the fruit is enclosed, together with the water and mineral substance brought up as food from the roots, and the starch made by the leaves and green fruit. As the fruit ripens the starch is converted into sugar. Passing from the twig into the fruit, by way of the fruit stem, are large numbers of sap-tubes which, as they enter the base of the fruit, separate, and, branching, permeate

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the flesh in all directions. These sap-tubes are the channels through which water and mineral food from the soil are carried to the living cells of the fruit. The apple is enclosed in a smooth, water-proof skin made up of a layer of cells, the outer walls of which are thick with a waxy infiltration of a water-proof nature. Scattered here and there, but most abundant toward the blossom-end of the apple, are minute openings through the skin known as lenticels. They are visible to the naked eye on certain varieties as minute brown specks. Through these lenticels air passes into and out of the apple; and water brought from the roots passes out as vapor. This process of water elimination is known as transpiration. Great quantities of water pass out in this way if conditions are favorable. Low humidity, high temperature, rapid movement of the wind, and intense light facilitate the process. If plenty of water is supplied by the roots, the process of transpiration removes the great bulk of it as vapor through the lenticels. If the supply of water is deficient, but at the same time conditions favor transpiration, then too much water is removed and wilting results.

When the brown spots are examined with a compound microscope it is found that the affected cells are collapsed or perhaps broken, and hence the tissue sinks, forming a pit. Whether the cells are merely collapsed or are broken is a question yet undecided. The cell contents are lacking for the most part, only starch grains, which were not converted into sugar during the ripening process, remain. Their presence is regarded as proof that the injury to the cells occurred before the ripening of the fruit. The cells are brown and have every evidence of being dead. Their brown color and their lack of sap, and therefore their dry texture, give the described appearance to the whole diseased area. Careful search will show that the brown spots always arise in close connection with a branch of the sap-tube system.

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With these facts in mind the most important theories, which have been championed to explain the killing and drying out of the affected cells, may now be examined.

(1) The injury is due to a rapid transpiration, or loss of water, from the cells, thus bringing about a concentration of the sap in those cells nearest the sap-tubes. This concentration of sap which is chiefly a concentration of the acids, results in the injury and death of the cells. The concentration of the cell-sap is therefore the immediate cause as outlined by this theory, and this concentration is increased by an insufficient water supply as well as by excessive loss of water. Those who cling to this theory believe that it accounts for the development of the disease both in storage and on the trees, and in the latter case for its appearance in wet as well as in dry weather. The following facts form the basis for this proposed explanation: (*a*) Warm, dry weather favors transpiration. (*b*) During such weather the soil is deplete in moisture and therefore cannot supply the cells with water sufficient to offset the rapid loss by transpiration. (*c*) During the wet season the apple-fruit grows very rapidly and the net-work of sap-tubes is not able to keep pace with the growth of the pulp-cells and the proper balance is disturbed. The sap-tube system is then deficient and the cells suffer from a lack of water. (*d*) The disease is favored by alternating wet and dry weather. Fluctuating temperature and humidity near the ripening period are highly favorable to bitter-pit, and conversely as long as the water is regularly supplied to the fruit, or at least as quickly as it is lost by transpiration, there is no pitting. Dry weather suddenly followed by heavy rainfall results in the development of stippen on the tree. And excessive transpiration, such as occurs in warm, dry weather, with a light crop of fruit is favorable to the disease. In other words, when transpiration is relatively greater than the water supply, stippen is produced. It is seen then that the weather conditions play a prominent rôle

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in causing this disease while the fruit is still on the tree. On the other hand, the methods of orchard management are contributing factors not to be overlooked. Heavy pruning is thought to favor the disease, for pruning affects transpiration. When the balance between the portions of a tree above ground and the roots under ground is disturbed, injury is likely to result. The application of manures not only supplies food but modifies transpiration. Green-manures conserve moisture in the soil. Manures of any kind invigorate the tree and enable it to use water from the soil in a more economical fashion. Cultivation conserves moisture and affects transpiration in a manner favorable to the tree. Irrigation must be taken into account. Regular and continuous growth is inimical to the development of bitter-pit. But if water is applied through irrigation in an injudicious fashion, conditions are made favorable to the disease. The disease also develops in storage. It has been found that fruit apparently clean when picked will show stippen after a considerable period in storage. Conditions in storage favorable to the disease are similar to those favoring it on the tree. Fluctuating temperature and humidity combined are evidently responsible for the production of bitter-pit. On the other hand, a uniform, low temperature and a dry air are unfavorable to the disease.

(2) The second theory brought forward to explain the cause of stippen is that the injury is due to excessive transpiration of water from the fruit during warm days followed by reduced transpiration during the cool nights. A warm day results in rapid absorption of water by the roots and in its rapid loss by transpiration. The ensuing cool night checks the elimination process, but the soil remaining warm allows the roots to go on pumping water into the tissues of the fruit. This results in an internal pressure which finally bursts the distended cells. They soon die and turn brown, and give rise to the symptoms of stippen.

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(3) A third theory is based on the assumption that the disease is most destructive in dry seasons. The premature dryness of the soil results in a failure of sufficient mineral food to reach the fruit, in consequence of which certain groups of cells starve and perish.

Many other causes have been assigned to stippen. Among the more prominent ones may be noted: local poisoning by spray material, poisons absorbed by the roots, bacteria, fungi, insects, mechanical injury, unfavorable grafting, degeneration from old age, and finally a peculiarity of the variety. A discussion of these various factors as causes of bitter-pit seems out of place in the present state of knowledge regarding the disease. None of them is known to be either direct or contributory causes of stippen.

Control of bitter-pit.

From the foregoing statements the following facts regarding bitter-pit are to be considered in discussing control:

(1) The disease has a very general geographical range.

(2) Some varieties of apples are more susceptible than others, although probably none is immune.

(3) The history of the disease indicates that it frequently becomes a serious problem to the apple-grower.

(4) The fruit only is affected and it usually shows the disease at the approach of maturity on the tree, or in storage.

(5) The disease develops beneath the skin at points near the sap tubes. This close proximity has suggested to the minds of those investigating the disease that stippen is in some way connected with the water supply of the fruit. Prominent authorities agree that a deficiency in the water-supply gives rise to the difficulty. Either the water is lacking in the soil, or it is eliminated from the fruit before certain cells have received their necessary quota. This results in sap concentration, chiefly an increased acidity within the cell, and the living substance is killed.

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(6) Factors having to do with transpiration are concerned in the cause of stippen.

(7) Intermittent weather conditions favor the development of the disease. A light crop of poorly distributed fruit is most liable to bitter-pit.

(8) Fluctuating temperature and humidity in storage favors the disease. Uniform, low temperature and dry air are inimical to it.

It is then apparent that growers should everywhere be on guard against this disease. No one can safely assume that his fruit will always escape by virtue of the variety which he grows or of the locality in which it is produced. It is very important that everything possible be done which will tend toward the production of good crops evenly distributed over the tree year after year; for herein lies a possible solution of the control of stippen. Most growers know that vegetative growth is unfavorable to fruitfulness. On the other hand, injudicious, heavy pruning to eliminate the vegetative growth also favors bitter-pit. It is believed that above all other orchard operations pruning has the most direct effect on the development of stippen. For it may regulate the amount, and to a certain extent the size, and it may determine the distribution of the fruit on the tree. The system adopted should be carefully planned, it should aim at the production of a regular crop of evenly distributed fruit, and should therefore be light. The lateral system of pruning, that is, the retention of laterals, as far as practicable, is to be adopted as a part of the general pruning-scheme, particularly in the case of varieties most susceptible to the disease. For there is some evidence that bitter-pit is much reduced on trees when the fruit is borne on lateral rather than on main branches. Frost at setting is also unfavorable to fruitfulness, therefore the question of frost-protection should receive attention in localities where experience has proved its necessity. Regulated sap-flow favors fruitfulness,

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and is also unfavorable to bitter-pit. Most alert commercial fruit growers practice thinning. It is an operation essential to regularity of bearing. It makes yields regular and bitter-pit is consequently reduced. Trees with a light crop and unusually large-sized fruit are most subject to pit. Again, overcrowding favors the disease. Manuring has a bearing on the development of stippen. The practice is commendable in that it not only supplies food, but neutralizes toxic substances in the soil. It also affects the moisture content of the soil and thus transpiration is modified. The water-elimination process in its relation to bitter-pit has been emphasized. Although long years of experimentation are necessary to determine the effect, if green-manuring were practiced in non-irrigated orchards the disease would probably be reduced. Cover-crops are planted by many of the leading apple-growers. These crops aid in regulating the water-supply of the apple, and when plowed under will conserve the soil moisture. The question of drainage should not be overlooked. This disposes of excessive water during the days of heavy rainfall, and conserves moisture during drought-periods. The disease is worst in low, wet portions of an undrained orchard. Moderation should be practiced in the matter of irrigation. The heavy application of water following a drought-period is favorable to stippen development. Excess should be avoided, and the aim should be to maintain uniformity of moisture conditions. Thorough orchard cultivation is advised. This operation affords proper aëration of the soil and consequent normal and efficient root-action. It conserves moisture during periods of dry weather, and as a result transpiration is properly regulated. Spraying or dusting operations have an important bearing on the problem; for, if neglected, the foliage is almost sure to be diseased and therefore less efficient both in the manufacture of food and in transpiration. It has been found that diseased leaves transpire less than healthy ones.

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The prevention of stippen in transit or in storage is relatively simple. Shipments should be made under cool conditions. The temperatures must be low (30 to 32 degrees Fahr.) and constant. In storage the same uniform, low temperatures must be provided. It has been shown conclusively that the development of stippen may be prevented by storing the apples at 30 to 32 degrees Fahr. under dry air conditions. The ordinary cellar or cool storage is not reliable. An increase in the temperature to 34 degrees Fahr. allows the disease to develop. The storage room should be properly ventilated; good ventilation in conjunction with refrigeration is of prime importance for successful storage of fruit.

It is thus evident that those practices which tend to a uniform, normal growth throughout the season, with fewest sudden stimulations or checks on growth, are, in the long run, most inimical to bitter-pit.

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Frost-Injury

Caused by the action of low temperatures

The remarks which follow apply for the most part to that phase of frost-injury commonly known as winter-injury. Properly winter-injury includes injury to all parts of the tree caused by low temperatures in the winter months. Some attention will be given to injuries due to spring frosts, but the general statements are made with a view to an explanation of the important problem of winter-injury. Of all the phases of frost-injury, sun-scald and crown-rot, or collar-rot, are most prominent and will therefore receive proportionate attention. Sun-scald is now regarded as a late form of winter-injury, while crown-rot is an early form of winter-injury. The term frost-injury is used broadly to include all injuries to fruit-trees due to the action of low temperatures without regard to the season of the year.

For many years it has been commonly observed that most fruit-trees suffer from the effects of severe cold and of sudden and extreme temperature changes in the cooler seasons. Authenticated records date back for a century and unquestionably the trouble was as common then as it is now. Theories were advanced immediately in earlier times to explain the occurrence and action of frost, some of which are still prominent, although each has been modified in some degree. All kinds of fruits are injured at some time or other; however, there is noticeable difference among fruits in this respect. It is generally accepted, for example, that while the apple is severely injured by cold, the peach is the more susceptible.

In the northeastern United States such apple-varieties as the Ben Davis, Northern Spy, Baldwin, Rhode Island and Tompkins King are all likely to suffer from cold. Others, such as the Oldenburg, Grimes and Hubbardston, often exhibit

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signs of injury from low temperatures. But these varieties are not invariably injured; and on the other hand those varieties omitted from the list are not always free from the trouble. Frost-injury seems to be confined largely to orchards between the ages of eight and thirty years. High-headed, severely pruned individuals of any variety on wind-exposed locations are most liable to the difficulty. Therefore, factors other than the nature of the variety condition the injury under consideration.

The rôle of frost-injury in fruit crop-production is a prominent and important one. Growers of fruits in practically all temperate and even in semi-tropical regions know and fear this trouble. But not every one appreciates the actual damage done in each case. It is only after hard disastrous winters such as were experienced in the East in 1903–1904, 1904–1905 and 1906–1907 that general complaints and appeals for help are made. Trees are severely injured and often killed in the extremes of winter temperatures of the northerly sections. In warmer regions late frosts sometimes entail heavy losses.

Symptoms.

Frost-injury involves all parts of the tree. Injury to aërial parts only are evident to the casual observer. A reliable symptom of fatal injury evident at the end of winter is not definitely known. Trees may appear entirely healthy, but on cutting into the bark above the snow line discoloration in the cambial region will be found. Trees, especially in low spots, affected by frost fail to start in the spring or they may make only a feeble growth. As the season advances they look unhealthy, the foliage is yellow, and finally death of the whole tree ensues (Fig. 8). Often a single branch or one side of a tree only is afflicted. Such trees on closer examination show frost-injured areas or cankers at one or more places (Fig. 9).

Injuries may occur chiefly on the southwest side of trees at a point midway between the crown and head, or they may be

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found at any place on any or all sides. Injuries may occur at any height on a tree; at the crown, the injury known as crown-rot or collar-rot; at the crotch, crotch-injury; on the main limbs as they diverge from the crotch, known as sun-scald; at the roots, root-injury. The tips of branches are sometimes killed back for some distance. Not infrequently the injury may extend from the collar to the head, involving large areas.

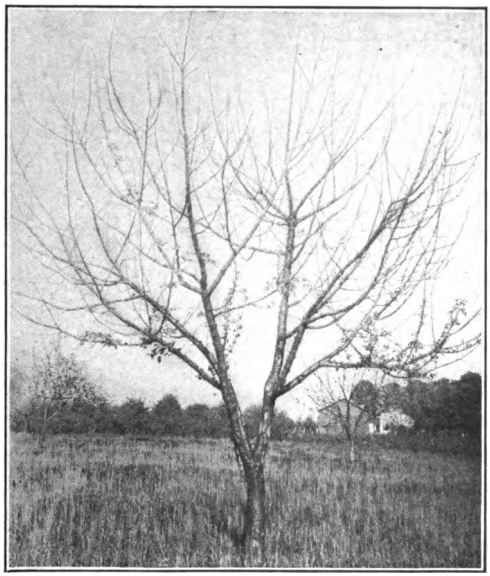

FIG. 8. — Apple-tree nearly dead as a result of low temperature injury.

Crown-rot, or collar-rot, occurs at the crown or base of the trunk at the ground line. It is distinguished from sun-scald and crotch-injury by its position on the tree. It is normally an early form of winter-injury. Observations indicate that it may occur from October to December in the latitude of New York, Ohio and Connecticut. The Tompkins King, Grimes and Hubbardston are most subject to crown-rot. In such injured places the bark appears discolored, dead and loosened from the trunk, sometimes split open, exposing the wood. Again the injured bark clings to the wood for a time, forming a canker at the crown.

Sun-scald is usually made evident in the late spring by the

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death of the bark on the southwest or sun-exposed side of a tree. The frosted bark may peel, exposing the discolored sapwood; or sometimes it adheres closely to the wood and a sunken, cankered area is thus formed. In some cases the sapwood is killed and the cambium is left alive to form new wood outside the dead area. The sapwood is stained by the diffusion into it of some substance apparently originating in the protoplasm of the affected tissues. When the wood is thus discolored it is called black-heart. This type of injury is common in the northern United States. Frost-killed areas in the bark are commonly inhabited by saprophytic and weakly parasitic organisms, prominent among which is the New York apple-tree canker fungus, *Physalospora Cydoniæ*.

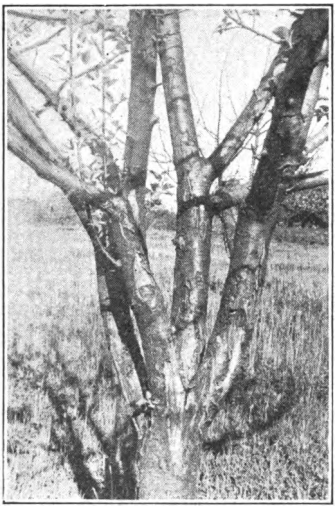

FIG. 9. — Apple-tree showing frost-cankers at crotch.

The name sun-scald owes its origin to the common belief that it is due to some interaction of sun and cold on the sunny side of the tree in late winter. It involves the crotches and the adjoining sun-exposed bark of the trunk and limbs as well. The Ben Davis, Stark and Twenty Ounce varieties are generally regarded as most susceptible to sun-scald. Trees leaning to the northeast are most severely injured.

Twigs are killed by winter temperatures. The last season's growth is killed back to a definite point. This occurs every year to some extent. It depends largely on whether or not the wood ripens properly in the fall.

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Roots suffer when the temperature is unusually low and the ground is bare. In some of the western states this is common and serious. It occurs more often on trees up to twenty years of age. The whole root-system may be killed, and the injury or death of tissues may extend to the crown. Ben Davis, Northern Spy and Wealthy suffer especially. Trees on crab-stock are said to be affected less than others.

The above described injuries on the roots, crown, trunk, crotch and branches are regarded as a winter type of frost-injury. Late spring frosts are sometimes troublesome, and do damage to the fruit-buds, blossoms, leaves and young fruits. All parts of a bud are not necessarily killed. Generally only the floral parts are involved, so that the buds will open in the spring and the killing will not be readily observed. Injury to the blossoms is a common form of frost-injury. Affected blossoms turn brown and die. It is likely that growers sometimes confuse loss of blossoms due to low temperatures with that caused by poor pollination. Young leaves are killed or injured at higher temperature than are old, mature leaves. Affected leaves crimp and curl; their upper surfaces are wrinkled and puckered and resemble peach leaf-curl to a certain extent. Heavy frosts at a time when the leaves are partially unfolded bring about the injury. Frost bands on young fruits are familiar to all. Occasionally late frosts occur which do not destroy the set of fruit, but there results a peculiar russeting. In older apples, this appears as a band of varying width entirely around the fruit midway between the stem and calyx-ends.

Cause of frost-injury.

It is held by earlier authorities that the various forms of frost-injury arise in different ways. That is, it is thought that crown-rot and sun-scald are different. However, it is now generally agreed that crown-rot and sun-scald are essentially the same, differing only in appearance and in location on the

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tree. It may be assumed, then, that irrespective of the time of the injury or of the organ affected, low temperature acts the same in bringing about such injuries. The explanation of the action of frost in the production of injury is based on the principle that there is a limit to low-temperature endurance by the plant. When this limit is reached or exceeded, the part is frozen to death or is seriously injured. This phenomenon is essentially a desiccation-process. The plant cell contains, on the average, about 75 per cent water which is necessary to the life of the protoplasm. The permanent removal of water from the cell, which occurs in freezing, is detrimental and the effect is expressed in serious injury or death.

A consideration of the cause of frost-injury involves a disposition of the many phases of the question of low temperature-action. To state that a given low temperature freezes certain tissues to death does not explain the manner of freezing. Among the more important phases in the process of injury the following may be noted: ice-formation in the tissues; slow *versus* rapid freezing; slow *versus* rapid thawing; bark tension and shrinkage of bark from wood; effect of snow and ice on the bark; alternate high and low temperatures.

It has been held in the past that ice-formation in the tissues takes place during the freezing-process. A warm, moist autumn, for example, offers conditions for excess water in the various tissues concerned. If winter should close in rather abruptly, the water in these parts would be frozen by the first period of low temperature. It was further believed that the formation of ice crystals in the tissues resulted in a tearing and rupturing of the cell walls. That ice is formed between the cells there is no doubt. Some, however, maintain that it is formed only when the freezing-process is very rapid. During such a process, it is thought, the water of the cell is withdrawn rapidly to the intercellular spaces; this rapid withdrawal of water from the cells results harmfully to them. At present the

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general opinion is that while the removal of water from the cells does act detrimentally, ice-formation itself is not directly responsible for the injury; it is only incidental and secondary.

The rate of the freezing has been a point of attention by many students of winter-injury. Prominent writers are agreed, however, that it is the sudden or rapid temperature-fall that brings about the injury. But this is not the final cause of the injury or death of tissues. During the downward jump of the temperature, water is rapidly removed from the cell, and death follows. On a cold sunny day tissues exposed to the sun are warmed, while tissues not exposed remain at or near the temperature of the atmosphere. Toward evening with the withdrawal of the sun's rays the temperature of the sun-exposed area falls so rapidly the first part of the period as to produce death of the tissues on the southwest side of the trunk, while that on the northeast side, protected by the slower temperature-fall, remains uninjured. Thus, in brief, the uniform presence of frost-injury on the southwest side of trees is explained. Localization of the injury at certain points, as, for example, the crown or the crotch, is due to the fact that the tissues at such places become mature more slowly than at other points. Perhaps, too, bark-tensions set up by rapid growth at these certain points also assist in localizing the injuries. And finally, rapid evaporation due to high winds may be a contributory factor.

Sudden thawing of frozen tissue is thought by some to be harmful in effect. In fact, one of the earlier theories advanced to explain the cause of sun-scald is that rapid thawing, subsequent to extreme temperatures, brought about by the action of the sun, produces the injury. This theory still has adherents. Those who hold to this explanation believe that during a rapid thawing the plant cell is unable to recover the water which has been removed during the freezing-process, and as a result the cell dries out and dies. But there are authorities who take

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the opposite view of the question and hold that rapid thawing is of no concern.

The question of crotch-injury is often explained on the basis that snow and ice lodge therein, soften the bark on melting, and injury results. Here, it is maintained, the sun's rays are concentrated through the water acting as a lens and the intense heat injures the protoplasm.

It has been suggested that possibly tissues become more tender as a result of frequent alternation of freezing and thawing. While sudden changes in temperature are destructive in effect, yet it is generally agreed that the sudden fall is of vital importance but that the injury is independent of the rate of thawing.

In general, then, whatever the manner of killing, it may be said that when, during the freezing-process, the water withdrawal from the cell passes a certain limit, the cell is killed. Some lay emphasis on the effect of the rate of freezing and thawing; others lay emphasis on the rôle of winds, snow and ice in assisting the action of low temperatures to bring about the injurious effects.

Frost-cankers, irrespective of their position on the aërial parts of the tree, are commonly inhabited by various fungi. The most common of these perhaps in the northeastern United States is the New York apple-canker fungus, *Physalospora Cydoniæ*. This fungus may live saprophytically, or it may assume the rôle of a facultative parasite. In the latter case it enlarges the wound and does considerable damage.

The topography of the land is influential in its relation to winter-injury. Trees on low, and consequently less-drained, land suffer more than others. The amount of water in the tissues at the time the low temperatures occur is a factor. The more water the cells contain, beyond certain limits, the more liable is the tissue to injury by freezing. On the other hand it is held that trees which have endured a drought during the

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growing season, will, if a severe winter follows, be seriously affected by low temperatures. A dry summer followed by a warm, wet fall makes a tree more susceptible to cold than it otherwise would be. Late cultivation and excessive nitrogenous fertilization forcing late succulent growth tend to an unripened condition of the trees, rendering them susceptible to early winter-injury (crown-rot). Sun-scald, however, is not induced by these conditions. The topography, type of soil, and methods of orchard management are factors influencing the severity of frost-injury.

Control for frost-injury.

From the foregoing remarks and discussion it seems advisable to reiterate in discussing control the following points with reference to winter-injury: (1) that while some varieties of apples are more susceptible to cold than others, none are immune; (2) all parts of a tree are liable to injury, and these injuries are referred to according to the part affected, as root-injury, crown-rot or collar-rot, black-heart, crotch-injury and sun-scald; these are forms of winter-injury, whereas spring frost affects the buds, blossoms and young fruits; (3) low temperature injury is essentially a desiccation-proccess, resulting chiefly from the sudden withdrawal of water from the cell during a sudden drop in temperature; (4) succulent immature tissues suffer more than properly matured tissues; (5) injured bark is inhabited by fungi which may enlarge the wound, preventing it from healing; (6) trees on low, wet soil suffer most; (7) a dry summer acts detrimentally, and if the autumn is wet, succulent tissue is developed; (8) injudicious nitrogenous fertilization and cultivation also favor winter-injury.

It is recommended: (1) that in planting, only those varieties best adapted to the particular soil and environment be selected. (2) Orchards should be cultivated thoroughly in the earlier part of the season, but the operation should cease in time to allow trees to mature thoroughly. (3) A cover-crop should

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be planted to take care of the excess moisture in the autumn. Such a crop would be particularly desirable following a dry summer. (4) Where irrigation is practiced, the operation should be discontinued in time to allow maturation. (5) The heavy application of nitrogenous fertilizers should be avoided except early in spring. (6) Low, wet soils should be thoroughly tile-drained. (7) Trunks and limbs of trees susceptible to sun-scald should be sprayed or painted with whitewash in the fall or early winter. This method seems more feasible than shading with a board.

But with all these precautions some injury will doubtless occur. Where frost cankers are developed, the injured bark should be removed and the wound treated as described on page 54.

The protection of the buds and blossoms from the action of spring frosts has been shown to be practicable. Orchard-heating is entirely possible and is practiced with profit in the West, and it is recommended for other regions. To combat frost, considerable preparation is necessary and the initial investment is large. The average cost an acre each year for heating is from $10 to $12. Materials such as coal, wood, crude oil, straw or rubbish of any kind are used as fuel. The weather conditions must be thoroughly understood in order to carry on the work successfully.

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Black-Rot Canker

Caused by *Physalospora Cydoniæ* Arnaud

This disease is best known as the New York apple-tree canker, in those regions bordering the Great Lakes, because of the prominence of this form of the trouble in that section of the country. In New England, Virginia and the Ozark portions of the United States, black-rot of the fruit and spots of the foliage are the more common forms with which the grower has to contend. Speaking generally for northeastern America, the Middle West and southeastern Canada, this apple disease, in one or more of its three forms, stands second only to apple-scab in importance. In other regions where it occurs it attracts considerable attention, but is plainly of less consequence than bitter-rot or apple-blotch. In the Middle Atlantic states it is less important than apple-rust. The disease, while present in most European countries, does little damage outside of North America.

Affected fruit is rotted and, as in the case of apple-scab, apple-blotch and bitter-rot, is rendered practically worthless

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so far as the market value is concerned. In New England considerable damage is done in storage. In Kentucky, Indiana, Missouri, Illinois, Alabama and Massachusetts the fruit is often seriously affected in the orchard. In Massachusetts 80 to 90 per cent of the apple-rots are of this kind, while in the other states just referred to black-rot is, next to bitter-rot, the most common type of apple fruit-decay encountered in the orchard.

The losses incurred through leaf-infections depend on the extent of the infection, that is, the size and number of spots on each leaf. In New York, only mild cases occur and the injury is not appreciable. In New Hampshire, Virginia and the Ozark region defoliation often results from the attacks of this leaf-spot pathogene. The Ben Davis, Black Twig, Chenango, Baldwin, Rhode Island and Twenty Ounce are most susceptible to leaf-spot.

The damage done to limbs is rarely appreciated. The largest limbs of mature trees are most subject to this disease, and while to most orchardists the loss of these limbs seems momentous, a great many are inclined to forget the cost of growing such limbs to bearing age as well as the expense of treating the same when thus diseased. Whole trees are sometimes killed. A case is on record where the trees on thirty acres of an eighty-acre orchard were ruined, and those on the remaining fifty acres were rendered almost worthless. The dollar loss incurred by the black-rot canker would be difficult to estimate. However, reckoning the amount of fruit lost as a direct result of canker on bearing limbs is a simpler matter, and for New York State the figures representing the annual loss through this channel have been conservatively put at $750,000. In those apple-orchards bordering Lake Ontario, the Twenty Ounce variety is by far the most susceptible. But it is difficult to point to a variety which in general is second in this respect. The Esopus, Baldwin, Wagener, Rhode Island and Tompkins

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King are sometimes so grouped. A peculiar and interesting case was called to the authors' attention in a New York State orchard where old trees of the Baldwin variety had been grafted to Twenty Ounce some twenty years ago. The point of union is about midway distant between the crotch and the highest tip of the branches. The canker shows abundantly on the Twenty Ounce portions of the tree, but the Baldwin stock is entirely free from the disease. In most cases the disease extends down the Twenty Ounce bark to the point of union, there stopping very abruptly. This indicates that the Baldwin is at least not to be classed with the Twenty Ounce and others of the most susceptible kinds. In Ontario, Canada,

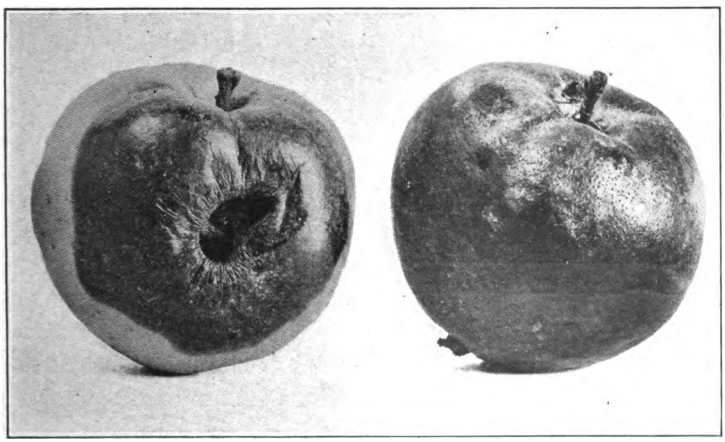

FIG. 10. — Early stages in the development of black-rot of apple.

the Ben Davis and Northern Spy are said to be susceptible varieties.

Symptoms.

Ripe fruits are more commonly attacked than green fruits. Signs of the disease may show anywhere on the surface or at the

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blossom-end. In the latter case, there is produced what is called blossom-end rot. Worm-holes are commonly the centers of rotted areas (Fig. 10). Ordinarily only one spot occurs in each fruit. Such a lesion is at first brown, and is frequently referred to as brown-rot. Very often concentric zones of light and dark colors of uniform width appear about the center of the lesion; in these cases the disease is called ring-rot. The margin of the diseased portion is distinct from that of the healthy (Fig. 10), and the rotted tissues are not of unpleasant taste as in the case of many fruit decays. Later stages of the disease exhibit a black color, whence the name black-rot. Usually by the time the spot is two inches in diameter small black fruiting bodies of the pathogene are seen on the lesion. Finally, a mummy is produced (Fig. 11), which is at first waxy, then dry and hard. Black-rot has been confused with brown-rot (Fig. 37, page 141), bitter-rot (Fig. 5, page 16), and soft-rot (Fig. 25, page 92). Brown-rot results in a browning of the affected tissues which is difficult in this stage to distinguish from black-rot. Later, the brown-rot mummy may be like the black-rot mummy only in that it is also black in the case of certain varieties; it differs, however, in that the brown-rot mummy is sometimes coal black, glossy, smooth, having no black fruiting bodies, and is much less wrinkled than the black-rot mummy. Bitter-rot, besides having an unpleasant taste, often shows pinkish spore masses on the lesion. (Compare Figs. 5, 10, and 11.) Soft-rot has an extremely softening effect on the tissues and also is attended by an offensive odor. These

FIG. 11. — Apple black-rot mummy.

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two characters serve to distinguish soft-rot from black-rot and other apple-rots.

The disease on the leaves is noticeable from a time shortly following their unfolding to the end of the summer. One to

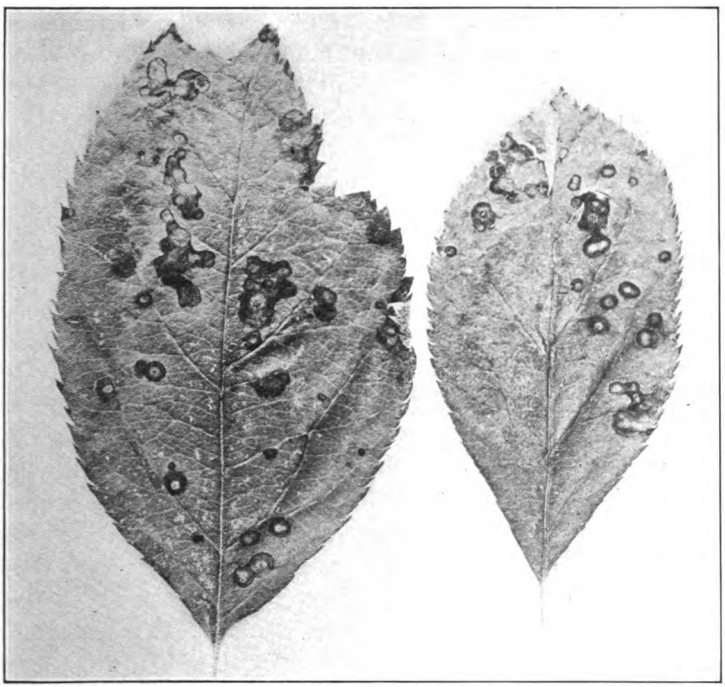

FIG. 12. — Leaf-spot of apple.

several spots occur on a single leaf; in the latter case the lesions are scattered or grouped (Fig. 12). On the upper surface the first evidence of this leaf-spot is a minute, purplish speck, which soon enlarges until it attains a diameter of about one-eighth of an inch. The spot later becomes yellowish brown, circular, and definite; in this stage the margin is elevated, and

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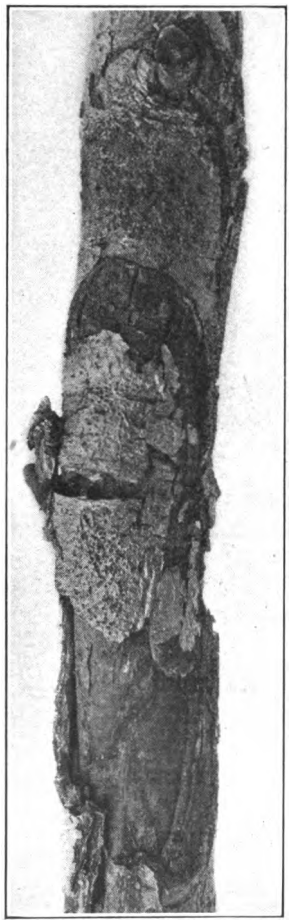

FIG. 13. — Black-rot canker.

the diseased portion is sunken. Older spots become lobed, due to the secondary extension of the pathogene from one or more points in the leaf. This activity results in a series of more or less concentric areas making up an irregular spot, but in which the outline of the original diseased portion can still be recognized (Fig. 12). The center of the lesion becomes grayish brown and the entire affected area presents an appearance which has given rise to the name frog-eye (Fig. 12). Frequently the small black fruiting bodies of the pathogene develop on the leaf-spot, although they are not always readily noticed on account of their minuteness. The spots are not so conspicuous on the lower surface of the leaf. In severe cases the foliage turns yellow and may fall six or eight weeks prematurely. Bordeaux injury is very similar to this leaf-spot.

The cankers (Fig. 13) are found more often on the uppermost side of the larger limbs. At first the bark is discolored and sunken. The discoloration is for a short time reddish brown but very soon is darker, the diseased portion, upon close examination, being easily distinguished from the surrounding healthy bark. Sometimes the cankers remain small and the pathogene dies at the end of the year. In other lesions the

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pathogene spreads from year to year until the canker is several feet in length. Very early in the formation of the canker a crevice is developed at its margin, on the healthy side of which corky tissue originates. This plate of cork limits temporarily the extent of the lesion. Further spread results in an increase of the diseased area with the formation of a second marginal crevice. Repetition of this process proceeds from one or more points at the edge of the canker until a series of more or less concentric crevices is developed (Fig. 13, near top). This phenomenon is very similar to that described for frog-eye of the leaves. Affected bark remains closely appressed to the wood for a year or more, but finally cracks and falls away, exposing the wood and a callus about the margin of the wound (Fig. 13). Before the bark falls there appears over its surface the same sort of black fruiting bodies of the pathogene as described for the other affected organs (Fig. 13). Girdling of affected limbs commonly occurs, as a result of which the parts above the lesion ultimately die. This is evidenced by a yellowing and browning of the leaves and the shriveling of the bark and fruit. Sometimes there is an hypertrophy of the limb at the upper and lower ends of the canker.

The above description constitutes external symptoms in which the bark appears to be the only part affected. But upon removal of the healthy bark immediately above and below the canker the sap wood is found to be stained brownish, the discoloration appearing in a long slender streak, continuous from the canker to a point several inches distant.

Cause of black-rot canker.

This disease is of fungous nature, and the causal pathogene is *Physalospora Cydoniæ*. The fungus passes the winter in the old cankers as mycelium and as pycnospores. It is not only found on fruit-trees other than the apple, but also on a number of other plants such as alder, ash, basswood, dogwood, elder, hawthorn, hop-hornbeam, lilac, maple, mul-

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berry, oak, pine, rose, sumac, witch-hazel and others. In the spring the pycnospores emerge from the pycnidia beginning in April; this discharge goes on throughout the growing-season. The spores ooze from their pycnidia in coil-like masses; the rain and wind carrying them to the leaves, fruits and bark. Insects are not known to disseminate the fungus. Germination of the spores occurs within six hours, the tubes entering the various susceptible parts through wounds, except in the case of young leaves which are penetrated through the uninjured surface. Within a few days symptoms of the disease appear as already described. Pycnidia, in which spores develop, are soon formed on the affected organs of the apple. These spores are carried to other susceptible organs throughout the growing-season. Some of the spores may be disseminated to any of the many plants previously listed. With the advent of winter the fungus ceases its activities, and lives through the dormant season in the form of mycelium and spores in the cankers. A perithecial stage is known, but it apparently plays no prominent rôle in the life-history of the pathogene.

Control.

The treatment of this disease consists in the eradication of cankers and in protecting the susceptible parts by the use of fungicides.

The treatment of cankers may be followed along one of two lines. The affected limb may be cut from the tree, or the diseased bark may be removed. The grower must study the problem and must proceed in accordance with the conditions. The size and value of the limb and the extent of the infection will be criteria. Small limbs, that is, those with a diameter less than one inch, should in all cases be removed. It would rarely be profitable to attempt to treat a canker on a branch of this size. If the larger cankered limb is valuable because of its producing powers, then the size of the canker must finally determine the course to pursue. Smaller cankers on large pro-

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ductive limbs can be profitably removed. If the lesion extends more than half around the limb, no attempt should be made at removing the canker; the entire branch should be removed.

In the removal of cankered limbs the cut may be made so that another shoot can grow in the approximate space left by the portion removed. The position of the canker on the limb determines the feasibility of the operation. In case the canker is at some point a few feet from the parent branch the affected portion of the limb may be removed by sawing off the same between the canker and the parent branch. On the cut end may then be grafted a desirable variety. This grafting of stubs is justified in certain cases, but should always be supplemented by careful spraying of the bark. The grafts should be watched closely, since the canker fungus may enter the limb at such points.

In cases where the whole limb is cut from the tree, the cut should be made at the junction of the limb with the parent branch. The cut should be made close to and perfectly even with the outline of the larger supporting limb without regard to the size of the resulting wound. A rough, splintered surface is highly undesirable and may be avoided by the following procedure preliminary to making the final cut. Saw about one-third through the limb from the lower side at a point one foot beyond the place where the final cut is to be made. The next cut on the upper side is made an inch beyond the first cut. The cutting on the upper side should continue until the limb is severed. This leaves a stub about a foot long which must now be removed, cutting, as previously outlined, close to the main limb. It is important that no stub be left, even if it is but a few inches in length, for the presence of a stub retards, or wholly prevents, healing. In those cases where a callus does not occlude the wound in a relatively short time, heart-rot is almost sure to result. It requires comparatively little time and expense to clean and

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protect a fresh injury or decay in its early stages. It often necessitates the expenditure of a great deal of time and money to treat properly the same injury after it has been neglected for a few years.

The cutting-out of cankers is a method to be employed when the orchardist is satisfied that the value of the limb warrants it. An attempt to remove all kinds and sizes of cankers from an infested orchard, without regarding the value of the limbs, is likely to result in discouragement with the whole matter. Such practice is neither good nor profitable. The grower's judgment must guide him.

Certain tools have been found advantageous for this work. A draw-shave for use in removing diseased bark, and a farrier's knife for trimming the margin of the wound, are the chief tools needed. The knife must be sharp, for a dull edge may injure the growing part. The pruner should not wear heavy leather-soled shoes, since canker fungi may get into the bark through wounds caused by such shoes. It is suggested that rubber boots, or some type of soft-soled shoes, be worn in connection with such operations.

In treating cankers it is necessary to determine the limits of the diseased tissue. This may be done by examining the canker externally or by shaving off bits of bark until the line of discoloration is located. The depth of the cut depends on the depth of any indication of disease, that is, discoloration. If the canker is for the most part superficial, penetrating the wood only in spots, the bark may be removed as described above, the deeper spots being rimmed out with a farrier's knife. If the fungus has entered the wood, either locally or in long streaks, the discolored part must be removed. If the streak extends for a considerable distance, the case may warrant the removal of the whole limb. So far as possible the wound when finally shaped should be pointed above and below, as this facilitates healing; if the cut is left in a rectangular form, the upper and lower

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edges heal more slowly. The edge of the wound should be cut at right angles to the surface of the bark; cuts made otherwise will result in a certain amount of dead bark, which makes an easy entrance for canker fungi.

It is a common, but false, belief that some substance may be applied to the surface of a wound to accelerate healing. No wound-dressing can induce more rapid callus formation, but it has been found very desirable and helpful to split the edge of the callus each spring, next to the wood, in order to stimulate wide spreading of the callus. The rapidity of the healing process depends on the character and position of the wound and the time of year when the wound is made, rather than on protective coverings. The sole object of coating a cut surface is to protect the heart-wood from decay until the new growth, which forms from the growing tissue immediately beneath the bark, has had time to develop over the exposed dead wood and protect it from decay. The fundamental requirements of a wound dressing, then, are that it be a preservative and a preventive. It should have antiseptic qualities, and should be fluid, reasonably inexpensive, and easily prepared and applied; it is essential that it give a complete covering; it must be impervious to air and water, must be durable, and must not injure nor kill the tissues nor interfere in any way with the healing process.

Preparations that meet all these requirements are not to be obtained. The substances most commonly used are paint, asphaltum and tars. Paint is an inefficient covering; asphaltum, once applied, gives good protection, but it is more difficult to prepare and to apply, especially when it is liquefied by heat. Asphaltum is more readily available if it is dissolved in gasoline. This combination has been used on apples with satisfactory results.

The writers have used coal-tar for the past three years with good success. This is produced in the manufacture of artificial

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gas from coal. It should not be called gas-tar, which is a term more loosely used. Some growers have complained of injury to trees from the use of coal-tar, but in such cases the material has usually proved to be something other than coal-tar.

The effectiveness of spraying for canker is a question frequently raised. As a preventive this operation is worthy of attention; as a cure it is out of the question, for once the fungus is in the bark the spray material cannot reach it.

If an orchardist desires to grow susceptible varieties, the canker menace may be obviated to a considerable extent by working over the larger limbs of more resistant varieties to the one desired. This has been done with apparent success in a few instances. The difficulties involved are that pruning must be done every year in order to remove all sprouts from the stocks, and that the renewal of old branches cannot be effected so rapidly. Such treatment also throws the bearing area higher in the air; so that in the case of erect-growing varieties such as the Twenty Ounce, it makes pruning, spraying and picking more difficult.

For the control of black-rot of apples, bordeaux mixture is said to be more effective than lime-sulfur. Apply bordeaux 4–4–50 as follows: (1) about July 15, when the disease is just appearing, and (2) two weeks later. As a matter of precaution against damage in storage, avoid injuring the fruit in any way. Spraying for leaf-spot should be done according to the schedule outlined for apple-scab (see page 12).

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Blotch

Caused by *Phyllosticta solitaria* E. and E.

This disease does its greatest damage in the central Mississippi Valley and is as well known in that section as bitter-rot. Unlike bitter-rot, however, apple-blotch is less sporadic in its nature, appearing from year to year and being gradual in its destruction. Over a long period, apple-blotch, because of its annual recurrence, is in the end more destructive than bitter-rot and apple-scab combined. This is particularly true in the Middle West and Southwest in such states as Arkansas, Kansas and the southern portions of Missouri, Illinois and Indiana. In northwestern Arkansas and southern Missouri 75 per cent of the apple-crop is commonly affected. It is almost as serious in the other regions indicated. In Benton County, Arkansas, the loss to apple-growers from blotch on the fruit in 1906 amounted to $950,000. The loss was about the same in 1907. In addition to rendering fruit unfit for market, the twigs are badly cankered and the badly affected foliage drops prematurely. The greatest loss occurs on unsprayed susceptible varieties. Among commercial apples which suffer most may be noted the Ben Davis, Missouri and Limbertwig; other varieties

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but slightly less susceptible are Smith Cider, Maiden Blush and others. The York Imperial, Winesap, Grimes and Jonathan are only slightly injured by blotch. In the East, blotch is rarely destructive and has never been reported from northeastern United States.

Apple-blotch, also known as the star-fungus, fruit-blotch, cancer, leaf-spot, and incorrectly as black-scab and late-scab, first attracted attention from Maryland and Texas in 1897. A few years later the same disease was reported from Illinois and in 1903 it was of serious extent in Missouri. It is said that the pathogene invaded Kansas about 1905.

Symptoms.

The disease first shows on the fruit (Fig. 14) about six or eight weeks after the blossoms fall. It is then evidenced by a very small, inconspicuous light-brown speck, which under slight magnification has the appearance of a stellate collection of brown fibers just beneath the epidermis. The spots enlarge radially, attaining a diameter of about one-fourth of an inch, and becoming darker in color. The advancing margin is irregular and has a fringed appearance (Fig. 14). The lesions show some interesting variations with varieties. Certain spots may or may not show a fringed margin, and may first be noticed as a dark, slightly sunken area. Not infrequently, the first indication of blotch is in the form of dark-brown, irregular, umbonate elevations. Such a condition occurs on the Ben

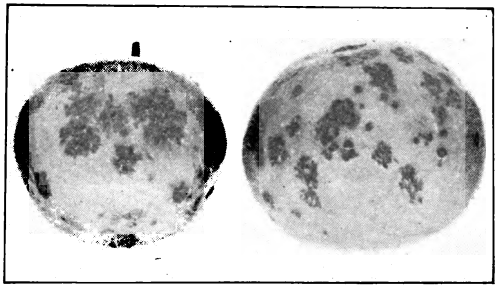

FIG. 14. — Apple-blotch, early stage.

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Davis. About the margin of older, sunken spots is sometimes seen a reddish border. On very young apples the points of infection show as small water-soaked areas, and in wet weather there may be a yellowish, gummy exudate. On some varieties at least, like the Ben Davis, the skin ultimately cracks (Fig. 15). The crevices, although usually about one-half of an inch long, may girdle the fruit and extend to the core. Often these cracks intersect, forming a cross (Fig. 15). Within a few days after the blotches become visible, black pimples are developed on the lesions (shown in Fig. 15). These are the fruiting bodies of the pathogene and they may number from three to several on each diseased area. They are either scattered promiscuously over the lesion, or are arranged around the margin. A large percentage of affected fruit drops prematurely.

FIG. 15. — Apple-blotch, late stage.

Apple-blotch also affects the fruit spurs, twigs and rapidly growing shoots, showing itself in the form of characteristic cankers (Fig. 16). Larger limbs are not commonly attacked. On the fruit-spurs the cankers are at first purplish or blackish. The center turns brown with age, the margin remaining unchanged in color. The resulting lesion is small and rather inconspicuous, with a crack along the margin. On water sprouts, and on other rapidly growing shoots, the cankers have much the same appearance as just described, but are longer, often measuring an inch or more in length, and sometimes girdling the stem. Longitudinal cracks appear not only along the edge

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but also through the cankered area, eventually giving the surface a roughened aspect. This is especially noticeable on cankers two or three years old. The diseased area may extend along the margin at several points, resulting in a lobed or somewhat concentric appearance. Cankers may or may not surround the limb; in the latter case the margin is marked by a crevice beneath which originates a callus.

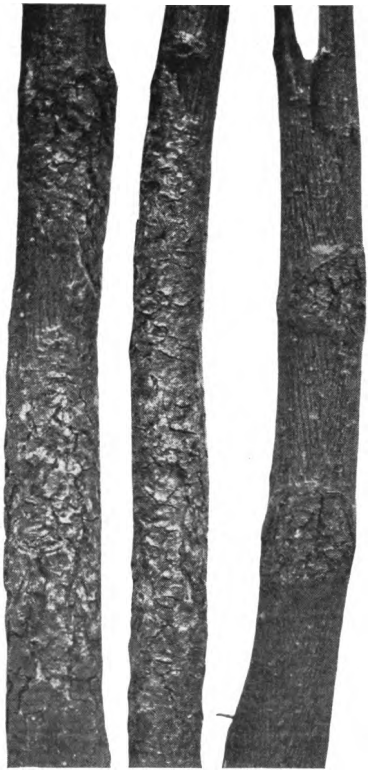

FIG. 16. — Apple-blotch cankers.

Affected leaves show spots within two months after the petals have fallen. The lesions are irregular in outline, light-brown to yellowish or whitish in color, and measure one-sixteenth of an inch, or less, in diameter. The spots are scattered indiscriminately over the leaf, occurring frequently on the veins, midrib and petiole. On account of their minuteness, several lesions may appear on a leaf without attracting attention, and perhaps without great injury. In more severe cases the diseased leaf is more noticeable and such foliage may be dropped prematurely. The leaf-petioles are attacked, in which case the leaves, instead of falling, turn brown, die and hang on the tree.

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Cause of blotch.

The fungus causing the various lesions just described has been known since 1895, at which time it was found on the leaves of wild crab; it was then named *Phyllosticta solitaria* E. & E. Two years later it was discovered on the apple, but not until 1907 was it known that the fungus on the apple was the same as that previously found on crab.

The pathogene hibernates in the cankers as mycelium. In the spring about the time the petals fall pycnidia appear along the margin of the lesion. Within three to six weeks, spores ooze forth from the pycnidia in great quantities, this period of spore-discharge extending from about the middle or last of May until the end of August. It is thought that wind, possibly insects, and certainly rain, act in carrying the spores from their spring quarters to the leaves, fruits and other twigs. Although the spores are disseminated as early as the middle of May, first signs of the disease do not ordinarily appear before some time in June. This means that approximately one month is consumed by the pathogene in getting established in its new environment. During this time the spores germinate, which process requires from twelve to eighteen hours; and assuming that moisture is present at the time of inoculation, then the remaining time is spent in developing mycelium and producing an injury of sufficient extent to become evident to the naked eye. The fungus grows superficially on the fruit, and in many cases the effect is not of serious nature beyond that of marring the appearance. On the other hand the attacks are often of a more detrimental character. Early in the development of lesions on the leaves, fruits and twigs, pycnidia appear. Spores are developed within these pycnidia on the fruits and twigs, although on the leaves the fruiting bodies seem to be sterile. However, the spores produced on the fruit and twigs are readily spread to other fruits, leaves and twigs. This phenomenon of secondary spread of the fungus appears to de-

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cline by the first of July, and no spores are disseminated after the last of August. After this season of the year the history of the pathogene on the leaves and fruits is unknown; but it is believed that these organs do not carry the fungus through the winter and hence are not sources of the first inoculum in the spring. It is on the twigs that the organism hibernates, a fact of great importance in dealing with the pathogene.

Control.

Since the disease characteristically occurs annually, control measures should be applied each year. There are two lines of remedial procedure: the removal of the cankered twigs and the protection of the susceptible parts by spraying. Careful pruning will remove a large portion of the diseased twigs, which are the source of trouble. Their removal is a valuable operation supplemental to the application of a protective spray. Spraying must be done before inoculation takes place; this, as has been seen, occurs within a month after the petals fall. The number of applications depends on the nature of the weather. In the Middle West and Southwest the schedule is as follows: First application, use bordeaux mixture, 3–4–50, three weeks after the blossoms drop. Lime-sulfur should be substituted for bordeaux mixture in wet weather, since the latter produces injury to the fruit and foliage under such conditions. Second application should be made two to four weeks after the first. A third application is recommended ten weeks after the petals fall. The second and third applications correspond to those made for bitter-rot, so that one course of spraying will suffice for both diseases.

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Rust

Caused by *Gymnosporangium Juniperi-virginianæ* Schw.

This disease is variously known as cedar-rust, cedar-rust of apple, cedar-apple, apple-rust and cedar-flowers. It affects not only the apple and wild crab, but also the red cedar. Both kinds of plants are necessary in the same immediate locality for the perpetuation of the rust-pathogene. If the cedar is absent, there can be no apple-rust.

With respect to the apple, a great many lists of susceptible and resistant varieties have been published from various parts of the country. These lists agree in part, but it is of interest to note that certain varieties are classed as susceptible in one state and as resistant in another. The Ben Davis, for example, is said to be resistant in Delaware, Massachusetts, Rhode Island and Nebraska, but is reported as being susceptible in Iowa and Virginia. The Rome is susceptible in Alabama, Indiana, West Virginia and other states, but is resistant in Delaware. Similar examples of such variation are found in the Grimes and Red June. While there may be some variation due to the difference in location, it appears that the judgment of the individual as to what constitutes resistance or susceptibility is a more probable explanation. Different periods of rust-infection may give rise to confusing data, since the leaves of one variety may expand more quickly or may have a shorter or longer period of susceptibility than the leaves of another variety. It has been found that varieties are susceptible only while the leaves are young; after maturity, they are immune. It has been learned also that the morphological make-up of the leaf, as, for example, hairiness, has nothing to do with the question of its susceptibility.

In spite of these uncertainties with respect to varietal resistance, it is generally agreed that the York Imperial is very susceptible. Others in this class are the Rome, Wealthy,

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Jonathan, Smith and Ben Davis. Among the more resistant varieties are the Baldwin, Yellow Newtown, Grimes, Arkansas, Maiden Blush, Stayman Winesap, Yellow Transparent and Winesap.

Apple-rust has been known to scientists for about a century. Important studies were made in New England about 1880. Since that date the rust has attracted more than usual attention in the states of Vermont, Iowa, Indiana, Alabama, Nebraska, West Virginia, Virginia, Kansas and North Carolina. The disease is now found to be widely distributed through the central and eastern portions of the United States.

The extent of the injury to apple-foliage is not always severe, but sometimes no less than 25 per cent of the leaves is taken from the tree. In many sections of the country the disease is serious on the fruits of the apple. The lesions not only mar the appearance of the fruit, but there is frequently a reduction in size and in quality. Rusted fruit is very likely to become infected with storage-rot pathogenes, thus entailing considerable loss. Rust itself does not induce trouble in storage. The red cedar is not only severely injured, but in extreme cases is killed. Where the tree is used for ornamental purposes this becomes an economic item. It is clear, then, that apple-rust may become important in one or more of the following ways: (1) by defoliating the apple; (2) by dwarfing and reducing the quality of the fruit; and (3) by incurring damage to the red cedar.

With respect to the amount of the losses resulting from this disease it may be pointed out that in 1912 the crop of York Imperials was an entire failure in many orchards of West Virginia. Actual fruit-losses ranging from $2000 to $3000 an orchard, due to rust, were very common throughout the eastern part of the state that season. The growers of one county lost $75,000. The disease is equally destructive in Virginia, Alabama, Iowa and Nebraska.

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Symptoms.

The presence of apple-rust in any locality may be detected by examining the apple or the cedar. On the apple leaves young spots may be found from April to June. At first these are seen on the upper surface as minute pale-yellow areas

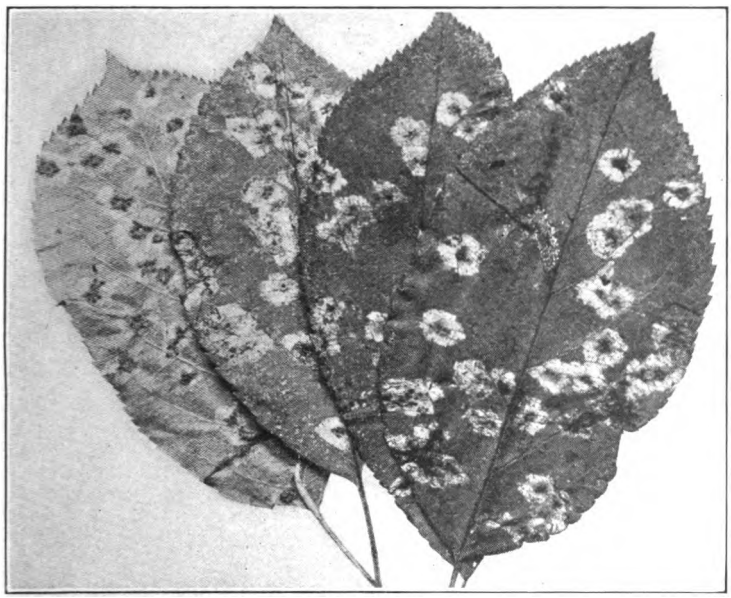

FIG. 17. — Apple-rust; on lower surface (on leaf at extreme left), and on upper surface (three leaves on right).

(Fig. 17, right). These enlarge, become darker, and finally are orange-colored. On the upper surface of the spots the fruiting bodies of the pathogene develop, first as minute, yellow, flask-shaped pustules, which finally turn black (Fig. 17). The leaf tissues beneath the spots become swollen and on the lower surface another kind of fruiting body is produced (Fig. 17, left). This one is cup-shaped and considerably larger than the

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one on the upper surface. At maturity the walls of the cup-shaped structures split and recurve in a stellate manner. In cases of severe infection the leaves turn yellow and fall. Sometimes 200 to 300 spots may occur on a single leaf. The diseased

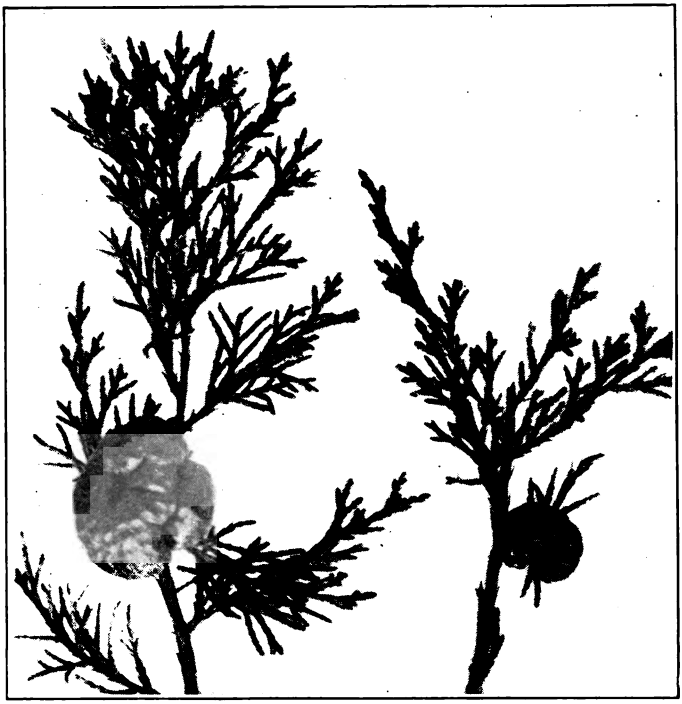

FIG. 18. — Cedar-apples in winter condition.

fruits are recognized by the presence of the cup-shaped fruiting bodies which are usually aggregated at the calyx-end. Not infrequently the lesions develop on the side or on the stem-end. As previously noted the fruits are dwarfed as a result of their being affected when they are young. Rust is rarely

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found on the woody parts of the apple, and then only on very susceptible varieties. Later in the summer small, greenish, spherical enlargements of the leaf may be observed on the cedar. Very soon these take on their final shape, which in some cases is reniform (Fig. 18). The enlargements or galls continue their growth, becoming brown and attaining a diameter of two inches or less by the end of the season. During the late autumn and early spring, these galls, or cedar-apples as they are called, show numerous depressions over their surfaces (Fig. 18) and in early spring a brown horn projects for an inch or more from each depression (Fig. 19). In rainy weather these horns become gelatinous and orange-colored. When thus fully formed the cedar-apple resembles a flower, whence the popular name cedar-flowers.

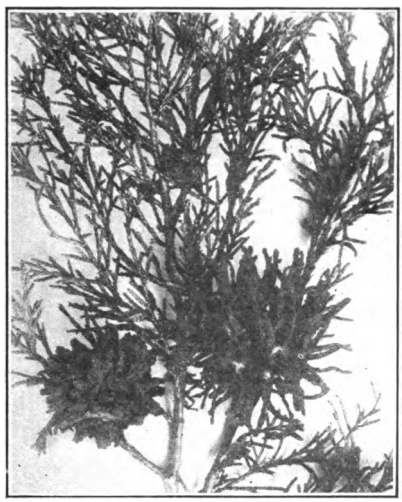

FIG. 19. — Cedar-apples in spring conditions. Note the horns.

Cause of apple-rust.

The apple-rust pathogene is a fungus, *Gymnosporangium Juniperi-virginianæ*, with a complex life-history as well as a long name. (The apple-rust fungus common in New England is *Gymnosporangium globosum* Farlow. This species occurs also on pear; see page 341.) Fortunately, however, the cycle of the parasite is well known to botanists and to many apple-growers. The fungus was first found and described on the red cedar, but no suspicion of its connection with the apple

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disease was then held. Several years elapsed before it was recognized that another stage of *G. Juniperi-virginianæ* occurred on the apple. Prior to this discovery the stage on the apple was referred to as *Ræstelia Pyrata*. After the heterœcious habit of the fungus was established, the last name was discarded and the former name was, and is now, used to designate it in all of its stages on the cedar and the apple.

In the spring basidiospores, or sporidia, are blown from the gelatinous horns of the cedar-apple to the young leaves and fruits of the apple. In some cases they may be carried several miles, but most of them do damage only at a mile or less. Under favorable conditions the sporidia germinate. Low temperatures, such as prevail in April and May, are most favorable to the germination of the sporidia. The germtubes penetrate the apple-leaf through the cuticle on the upper surface. The mycelium develops locally within the leaf, occupying the spaces between the cells and sending haustoria into the cells. As a result of the stimulative action of the mycelium, the leaf is hypertrophied, due to an excessive enlargement and multiplication of the cells in the lower part of the leaf (spongy parenchyma). These cells become elongated and the intercellular spaces of the spongy parenchyma are obliterated. The apple-fruit tissues are at first enlarged, but finally growth of the fruit at the point of attack is stopped, which fact explains the marked dwarfing and reduction in size. About one month after infection, the mycelium forms the first fruiting bodies of the season. These are on the upper surface of the leaf or on the fruit and are called pycnia (spermagonia). Spores are developed within the pycnia and are called pycnospores (spermatia); their function is unknown. They do not reproduce the fungus. Within a short time after the spermagonia appear, the cup-shaped bodies develop on the lower surface of the leaf opposite the spermagonia; these are known as æcia. About the middle of July these open and their spores, called æcio-

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spores, are discharged. The wind carries them to the red cedar where they cause the formation of the cedar-apple. It is not definitely known whether the æciospores germinate in the fall or in the spring, but at any rate mycelium develops from the germtube and the cedar leaf-tissues are stimulated as are those of the apple. As a result of this over-growth a gall is formed. This is the cedar-apple. It will be observed that one year elapses between the inoculation of the apple and the appearance of the gall on the cedar. This gall continues to grow throughout the summer and by fall has attained its full size. The next spring brown horns push out over the surface of the gall; these horns are composed of another kind of spores — teliospores. With the advent of spring rains the horns become gelatinous and the teliospores germinate. This occurs approximately two years after the first inoculation of the apple. A peculiar germtube is produced by the teliospores — one which is four-celled and which is called a promycelium. Four sporidia, or basidiospores, are developed on each promycelium; each sporidium is discharged at maturity and is blown to the apple. Thus the complete life-cycle involves a period of about two years.

The teliospores do not all germinate at once. As already noted, during a wet period they germinate and produce sporidia. If the air then becomes dry, the sporidia are snapped away and caught by the wind. With the return of rainy weather the process of gelatinization of the horns and germination of the teliospores is repeated. As evaporation occurs the second crop of sporidia is disseminated. In certain years there may be but one crop, in others there may be as many as six crops. The number and frequency of these yields of sporidia are determined by the weather conditions. The air must remain highly humid for at least six hours in order to allow gelatinization, germination, and sporidial production; but within this short period a great number of sporidia may be produced and with

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the return of dry air they are promptly carried to the apple. It is clear, then, that following a short rain it is but a question of a few hours before the fungus is actually inside the apple leaf.

The relation of the development of *G. Juniperi-virginianæ* to the weather is very striking, and is a question of great importance as well as of great interest. From what has been said it is obvious that the weather factors concerned are moisture, temperature and wind. Moisture is necessary for sporidial production. Winds and warmth aid in evaporation and in sporidial liberation. Moisture again is necessary for germination of sporidia, æciospores and teliospores. Temperature is likewise a limiting factor in this connection. Winds appear to be the only agents of inoculation. It is not necessary that the winds be strong, since the spores are very light and float easily in air currents.

Control.

The simplest and most effective method of handling apple-rust consists in the eradication of the red cedars. This is practiced by larger growers where the disease has become important. This work must be done thoroughly. All red cedars within a radius of at least one mile about the apple orchard must be destroyed. The cost of this work has been found to be less than forty-eight cents an acre in West Virginia. It has been found that galls may produce teliospores and sporidia two months after the trees are cut. It is therefore advisable to burn any red cedars which are cut later than March first. The removal of galls from the red cedars is a commendable practice only when the cedars are more desired than the apples. Such may be the case in public parks and on private estates. The galls should be removed in advance of sporidial production, that is, before March first, to make sure of effectiveness. The work must be done every year. Spraying the cedars likewise is to be done only where the cedars are

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few and highly valued on account of their ornamental qualities. In any case the value of the operation is as yet uncertain. Spraying apples to protect them against sporidial infection is unreliable. This is because effectiveness is so dependent upon the time of the application. A delay of one day after telial gelatinization makes spraying the apple of no value. The time in which effective spraying may be done is not long enough to allow for covering a large orchard efficaciously without undue outlay for machinery and labor. Where orchards are far enough from the cedars to escape severe infection and where only moderately susceptible varieties exist, the application of a fungicide may prove effective. Under such conditions spray the leaves as soon as they unfold and keep them protected until the first week in June. Subsequent applications should be made often enough to protect the new leaves as they appear. Lime-sulfur 1–40 may be used. Wherever cedars occur and it is not feasible to destroy them, the young orchards should be set to resistant varieties, avoiding particularly the York Imperial, Rome and Wealthy.

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White Heart-Rot

Caused by *Fomes igniarius* (Fries) Gillet

This disease is far more important in the forest than in the orchard. Among some of the trees attacked, besides the apple, are beech, aspen, balm-of-Gilead, willow, sugar maple, red maple, striped maple, silver maple, yellow birch, butternut, black walnut, oak and hickory. Most destruction is wrought in the beech and aspen. But as an apple-tree heart disease, white heart-rot is perhaps the most prevalent and the most destructive trouble of this type.

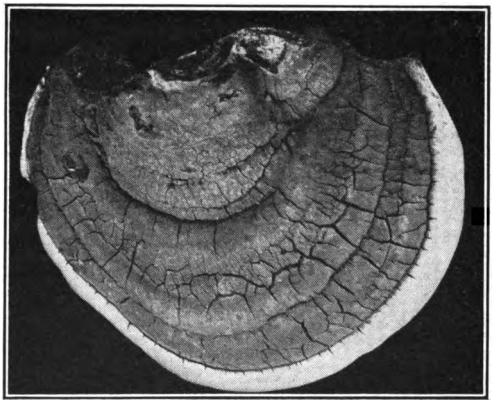

Fig. 20. — Fruiting body of the white heart-rot pathogene.

Although this disease has been known for about two hundred years, it was not thoroughly studied until 1878. Little has been learned since that time, in spite of the fact that the disease is world wide in its range. It has been found in practically every country of the globe as well as in all the more important islands. The causal pathogene does not appear to be limited in its geographical range by climatic conditions, being found not only in temperate zones, but in the frigid and tropical regions as well.

Symptoms.

The characters by which white heart-rot may be recognized are of two general types, external and internal. The first

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external evidence of the disease is the appearance of the characteristic sporophores of the parasite (Fig. 20) which grow out through knot-holes where branches have broken off (Fig. 21, right). These fruiting bodies are more or less hoof-shaped, hard, black, and checked on the upper surface (Fig. 20), and dark-brown and porous on the lower surface. The pores are extremely small, their diameters being not more than one-sixteenth of an inch. The size of the sporophores themselves varies greatly. The internal symptoms of disease are evident when the tree is cut or blown over. Cross sections of the diseased portion show symptoms quite distinctive of the trouble (Fig. 21). Affected trees never become hollow, but the rotten wood remains in place with a few cracks. The central area of the diseased heart is whitish or light-yellow (Fig. 21). Bordering this area is a narrow black line; sometimes there are several of these black lines arranged concentrically with white areas between them, and a yellowish to reddish brown zone, with an indefinite border, just outside the outermost black ring (Fig. 21). The character of the wood in these areas is as follows: — (1) in the white central area the wood is soft and crumbly when rubbed between the thumb and finger; (2) between the concentric black lines it is similar to that in the center, except that decomposition has not progressed so far; (3) outside the outer-

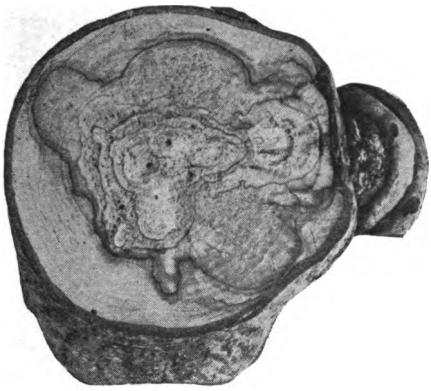

FIG. 21. — White heart-rot; cross section of an affected limb showing the rotted heartwood, the black lines and at the right a young fruit-body of the pathogene.

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most black line the wood is hard and firm and thus differs from the normal wood only in color. The brown discoloration is caused by decomposition products which diffuse into the bordering healthy tissue.

Cause of white heart-rot.

The causal pathogene, *Fomes igniarius*, is one of the pore-bearing basidiomycetous fungi. It has been known for about two centuries and was formerly used as tinder or touch-wood, or beaten into soft square pieces to be used by surgeons for stopping bleeding arteries. It is sometimes called the false tinder-fungus, the true tinder-fungus being a near relative (*Polyporus sulphureus*). Its life-history is similar to that of other basidiomycetes of this type. The spores, produced on the basidia lining the pores, are matured and disseminated during the early part of the summer. Some of these lodge in wounds where they germinate and, setting up a food relationship with the host, initiate the rot. The most common point of entrance is a knot-hole, or a stub exposed by careless pruning operations. From the germtube, mycelium is developed which grows into the heart-wood of the tree, passing up and down and obtaining the food necessary for its further growth. The rate of spread of the mycelium is dependent on many factors, as, for example, the breadth of the annular rings and environmental conditions. The rate is more constant in the horizontal than in the vertical growth. The mycelium develops abundantly in the wood-parenchyma, medullary-ray cells, and sometimes in the interior of the sap-tubes. It passes as a brown fungal mass from the sapwood into the bark. From here it presses outward and upon reaching a wound or bark fissure begins to form its sporophores. These originate by the massing of mycelial threads into a more or less definite form. On the lower surface of this mass a layer of tubes is formed; in each tube basidia and spores are developed. The spores break away, fall through the tube and out, are caught by the wind and carried to new infection-

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courts. The mycelium lives over in the tree from year to year and the sporophores live for several years. One case is recorded where the sporophore reached an age of eighty years. Each year the mycelium making the sporophore fills up the old tubes and builds on a new layer of pores just below the layer of the preceding year.

Control.

For the orchardist, it will be well to consider the possible source of the trouble. It has been pointed out that the disease may be found on a wide range of broad-leaved trees. Should the orchard stand in close proximity to the forest, more than the usual amount of trouble may be expected to ensue. In such an event the destruction of diseased forest trees and the removal of sporophores become important. Perhaps the most important matter after all is the avoidance of wounds. Pruning wounds should in no case be left uncovered; a wound-dressing should be applied within a short time after pruning operations. Coal-tar will be found entirely satisfactory.

The making and handling of cuts have been discussed on page 53. In cases where trees are already affected with heart-rot the treatment of such areas is an important matter in the control of the disease. It should be borne in mind that the normal heart of a tree is practically dead tissue. It gives only rigidity, and may be completely removed without infraction on the normal functions of the tree beyond impairing its strength, which is a serious consideration, however, because the limbs may be broken, or the tree may be blown over by the wind. Decaying wood can be of no use to a tree; on the other hand, it may act detrimentally and should therefore be removed. The operation should be thorough, all decayed wood being removed. A mallet, a chisel and a gouge are the chief tools needed. If the rot extends for a considerable distance down the limb, a hole, which will serve as a drain, should be bored at the lower extremity. Cavities in shade and orna-

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mental trees are often filled with cement or asphaltum blocks. Whether or not this practice is to be followed in the apple orchard depends upon the extent of the injury and the exposure of the orchard to wind. As a general rule the filling of cavities with any substance is not within the province of the practical orchardist; the operation does not pay. But in any case the interior surface of the cavity should be coated with a wound-dressing.

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BLISTER-CANKER

Caused by *Nummularia discreta* Tul.

This disease, which is known as blister-canker, and Illinois canker, was first given attention in Illinois in 1902. Since that time it has been reported from the following states: Arkansas, Indiana, Iowa, Kansas, Missouri, Ohio, Oklahoma, Nebraska, New Hampshire, New York, Virginia and West Virginia. Besides its occurrence in the United States, the blister-canker is found in Germany, Italy and Cuba, although it does not appear to have assumed great importance in these countries.

In certain states, as, for example, Missouri, and the southern portions of Illinois, Indiana and Ohio, this is the most injurious canker disease of the apple. It is said that within the last few years orchardists in Kansas, Missouri, Oklahoma and Nebraska have lost more than a million trees from it. The injury con-

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sists in the destruction of both large and small limbs. Not infrequently large trunks are affected, and trees are killed. The only available estimate upon the actual importance of this disease comes from Ohio. In the southern part of that state from 1 to 90 per cent of the trees are affected, amounting to an annual loss of thousands of dollars. In the summer of 1912 thousands of trees, and in many cases entire orchards, were practically ruined by blister-canker in Iowa. The Ben Davis appears to suffer more than other varieties.

FIG. 22. — Blister-canker of apple. Old canker, showing "nail-heads," or "blisters."

Symptoms.

The later stages of the blister-canker are very characteristic (Fig. 22). However, in the earlier stages the lesions are distinguished from those of other cankers with some difficulty. At first the spots are not conspicuous, and would be easily overlooked by the casual observer. The bark takes on a dingy, brown appearance, the size of the cankers varying at this early age. The lesion enlarges most rapidly in the direction of the long axis of the limb. The interior of the generally diseased area shows healthy areas of bark scattered through the whole affected portion. This gives a peculiar mottled appearance to the canker. The margin of the diseased bark is sharply delimited from

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the healthy tissues, sometimes a crevice marking the boundary of the canker. The dead area is usually a little depressed, due to a dying and shrinking of the bark. Frequently branches are girdled, hence the parts above wither and die. It is no uncommon sight to see dead fruits and leaves clinging to limbs which have just been girdled by the blister-canker pathogene. In late summer and autumn small tan-colored cushions, fruiting structures of the pathogene, appear under the bark at or near the margin of the canker for that season. Later the cankers become blackened and appear burned. The bark becomes very dry and brittle, and irregular patches fall, exposing the dead wood (Fig. 22, at the top). Sometimes whole trees are barked; and frequently one side of a tree is thus devoid of its bark, the other side remaining normal. Cankers sometimes extend from the roots to the limbs ten feet above ground. Ordinarily, however, the cankers extend three feet or less, and are confined to the larger limbs. Older cankers show, in place of the cushions mentioned above, dark fruiting cushions, called "nail-heads" by growers (Fig. 22). The presence of these cushions gives a blistered appearance to the canker, and they form the most pronounced distinguishing feature of this disease (Fig. 22).

Cause.

The pathogene, *Nummularia discreta*, was described from eastern America in 1834, but at that time it was not recognized as the causal factor in blister-canker. Now there is no doubt as to the ability of this fungus to cause this disease.

The fungus is in no strange habitat when found on dead wood. And it appears that it is unable to establish itself without the presence of an injury of some kind in the bark. It may live about the orchard indefinitely on dead limbs of the apple; it also easily lives on Sorbus, Cercis, magnolia and elm. Hence these trees may be regarded as possible sources of trouble wherever blister-canker prevails.

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On the surface of the old fruiting pustules — the stromata — ascospores can be seen in the spring exuding from the sunken perithecia. These spores are washed and blown to infection-courts, chiefly wounds, where they germinate and their germ-tubes establish a food relationship with the bark. It has been observed that one of the most common seats of blister-canker injury is in old stubs which have been left by careless pruning. The fungus now in the bark extends into the wood where it grows even more rapidly than in the bark. Some limbs, if carefully examined, show brown, discolored wood at points several feet away from the canker. It is probable that the fungus can gain an exit where the bark is injured, at which places new cankers would be formed. Wherever the mycelium penetrates the bark the tissue is killed, and finally is disintegrated. For some reason the spread of the fungus within a given area is not complete at first, but here and there sound bits of bark are left unaffected for a time. This explains the mottled appearance of the younger cankers. Finally, however, these spots are also killed. The growth and spread of the hyphæ within the bark is so rapid that no callus is laid down at the advancing margin as in many other canker diseases. The only attempt made on the part of the tree to inhibit the advance of the fungus is the formation in the summer of a layer of cork. It seems that growth of the pathogene in the tissues continues as long as there is sufficient oxygen and moisture present. Its attack on heart-wood and sapwood appears to be conditioned by these factors. The supply of air and water must be in proper proportions.

In July and August the fungus develops its stromata by a massing of the mycelium in certain portions of the diseased bark. As they increase in size the bark is ruptured, exposing their light, tan-colored surfaces. On these disc-shaped stromata are developed, on erect conidiophores, honey-colored conidia. It is believed that these conidia may be carried to

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the limbs of the apple and other hosts where infection results. Just when these conidia function as inoculum is not clear, although possibly as soon as developed. Whether they are capable of overwintering is not known.

As the stromata grow older a ring of black stromatic tissue is formed beneath the disc. The ring, or cylinder, extends into the wood of the limb. The connection of the stromata with the wood allows them to persist for as long as ten years, even after the bark has long since fallen away.

In the upper portion of the stromata there are formed, from April to June of the following year, flask-shaped cavities, with long necks opening at the surface; these are the perithecia. These contain asci and ascospores; the ascospores are discharged and come to lie in small, black heaps on the surfaces of the stromata. This expulsion is said to occur in the spring, but ascospores may be found on the discs at any time. It is probable that they cling there for a year or more; at least their vitality is retained for several months. There is some indication that the woolly aphis carries the spores.

Control.

The blister-canker is one of the most difficult diseases to combat. The rapid growth of the fungus in the heart-wood renders a cure practically impossible. If the canker is found in its early stages, however, it may be profitable to cut away the injured bark and wood and cover the wound with coal-tar or some good dressing. (For methods of cutting-out cankers, see page 54.) Limbs showing older and more extended cankered areas should be removed entirely. Since the fungus enters the tree through stubs left by careless pruning, a step toward the control of this disease consists in avoiding such indifferent procedure. It should always be remembered that the fungus can get into the bark only through injured tissue. In those regions where blister-canker is especially troublesome, resistant varieties may be sought with profit.

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NORTHWESTERN ANTHRACNOSE

Caused by *Neofabræa Malicorticis* (Cordley) Jackson

This is a disease peculiar to the Pacific Northwest. In consequence of this fact it is often called Pacific Coast canker and Northwestern apple-tree anthracnose. It is known to occur most abundantly in the states of Washington and Oregon. It is not recorded in California, although it probably is present in the northern part of that state. It was once reported as far east as Nebraska, but the disease is unknown in any section of the United States other than that already indicated. Outside of the United States it is found only in British Columbia. It is believed that the apple-tree anthracnose originated somewhere within its present geographical range on some native host. It did not begin to attract serious attention on the apple, however, until 1891. Since that time its ravages have increased until in the Northwest the anthracnose is second in importance only to apple-scab. It is most serious in those regions west of the Cascade Mountains, where considerable rainfall occurs. It is rarely serious in eastern Oregon.

On account of the nature of the injury the amount of the losses is difficult to estimate. The twigs, sometimes the larger branches, and even the trunks of young trees are likely to be girdled. A single tree may show from one to more than one hundred cankers varying in length from one-fourth to six

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inches or even larger. Affected branches are weakened and often are broken by a heavy load of fruit. Stored fruit and even fruits still in the orchard are rotted by the anthracnose pathogene.

Symptoms.

The disease is known by the cankers which are produced on the woody parts (Fig. 23). These are characteristically dark, sunken, dead areas in the bark. On this account the disease has been called black-spot canker, black-canker and dead-spot. Cankers are found most abundantly on the younger branches; that is, those having a diameter of two to three inches or less; but larger limbs and trunks are not always free from such lesions. On older branches the cankers are often more or less superficial, not extending to the wood. But ordinarily on younger limbs the bark and cambium are dead and the sapwood is discolored to a limited extent.

The first evidences of this canker appear ordinarily from November to January, depending on the season. The most numerous and the most destructive cankers appear in November and December. When first evident, the lesion is circular, less than an inch in diameter, and the surface is not sunken. In this stage the bark is discolored beneath the surface and has a water-soaked appearance. Soon the discoloration extends to the cambium and here it spreads out, often being more extensive than the surface discoloration would indicate. The canker develops very little through the winter, but in March and April it enlarges rapidly. As it enlarges its shape becomes ellipsoidal, and the surface sinks slowly. Sometimes the bark shows concentric zones of slightly varying color. The margin of the canker in the later stages of its development is limited by a crevice, which is finally bounded by a callus.

Toward the middle of the summer small, conical elevations are observed on the canker. These burst through the bark in a triangular or transverse manner, exposing a creamy mass

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— the fruiting structures of the pathogene. By late summer or fall the canker may have attained a depth of one-half an inch, a length of ten inches, and a breadth of three to four inches. Sometimes two or more cankers become confluent and thus larger lesions are produced. In older cankers, the bark may drop out leaving a wound, although this may not take place before the canker is three years old. The smaller cankers sometimes heal slowly by callus-formation. In other cases the wound never heals, but instead the limb is completely girdled.

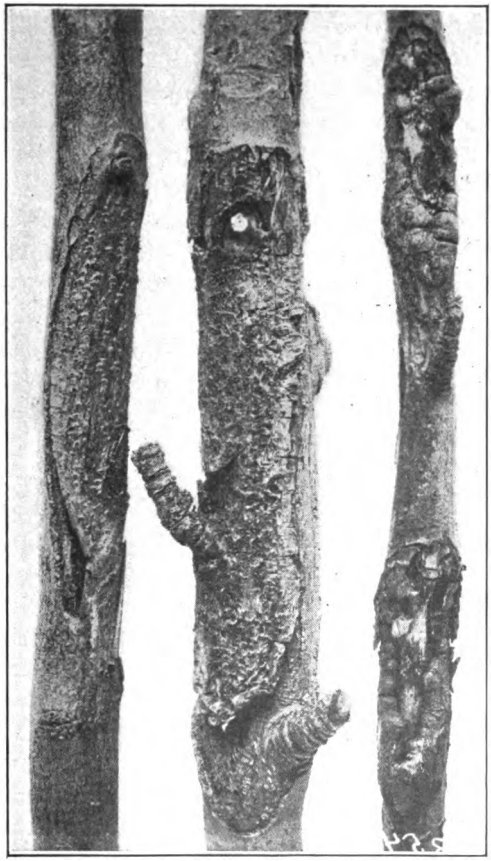

FIG. 23. — Northwestern anthracnose-cankers on apple-limbs.

The disease is not uncommon on the fruit, either in the orchard or in storage. The lesions may begin anywhere on the surface; frequently they center about one end, or about an injury of some sort. The

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flesh of the diseased portion becomes light-brown, the surface depressed, and the texture dry and leathery. Within the affected area the fruiting pustules of the pathogene develop in concentric circles; finally they split open, exposing the creamy substance as described for the canker.

No variety is wholly immune to the disease and some are nearly ruined by it. While it would appear that certain varieties are generally more susceptible than others, the degree of susceptibility varies considerably in different orchards of the same variety, and in different trees of the same variety in any given orchard. The evidence indicates that the kind of soil has little to do with susceptibility. Some hold that the Baldwin, Spitzenberg and Jonathan are most commonly attacked, and that the Newtown, Rhode Island and Gravenstein are somewhat less susceptible. Those showing less tendency to be affected are the Ben Davis, Northern Spy, Winesap, Tompkins King and Hubbardston.

Cause of anthracnose.

The apple-tree anthracnose is caused by the fungus *Neofabræa Malicorticis*. In its structure and habits it is very similar to the fungus *Pseudopeziza Ribis* Kleb., which causes the currant-anthracnose. It spends the winter as almost inactive mycelium in the cankers. In the spring the fungus renews activities for a short period, but usually stops its further spread as soon as the cambium becomes active. In midsummer those cankers which were initiated the previous fall form acervuli (Fig. 23, extreme left). With the advent of the autumn rains the acervuli become active, the conidia oozing forth in a creamy, gelatinous mass. On being dried by the wind the conidia are carried to other limbs and to fruits. With the return of the rains the conidial masses again ooze out, and the process of dissemination is repeated. In many cases conidia are doubtless washed by the rain to points below the cankers. In general the conditions prevailing from October to December are very

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favorable to spore-germination. The fungus is capable of entering its host through wounds in the bark and fruit; however, these wounds are not necessary to penetration. The uninjured bark is penetrated in most cases, chiefly through the lenticels. The healthy, uninjured skins of fruits may be penetrated directly. The fungus in the fruits produces the rot already described, but such fruits apparently never play any further part in assisting the fungus to complete its life-cycle. The fungus undoubtedly perishes with the destruction of the affected fruit. In the limbs, however, the fungus develops advantageously. It grows slowly after entrance into the bark, killing the bark-cells as it progresses. The cambium, sapwood, and even the heart-wood are also affected beneath the canker, but there is no evidence that new cankers arise by growth of the mycelium to points above or below such lesions. As previously pointed out, the fungus develops very slowly during the winter. The following March and April growth is resumed; growth ceases, however, with the initial spring activities of the cambium. By midsummer, the acervuli are produced, and the first year's cycle is completed.

In the fall, two years after infection, the sexual stage of the fungus develops in the old cankers (Fig. 23, center). The fruiting bodies formed are apothecia. They arise in the clefts in the bark formed by the old acervuli, developing in the mycelial cushions left by these asexual structures. Under the moist conditions of the autumn the apothecia become swollen; if dry weather prevails for a time, they seem to disappear, and then to reappear with the return of the next wet weather. If favorable conditions prevail for any length of time, the asci elongate, swell, and the ascospores are finally ejected forcibly. These spores are carried by the wind to the susceptible organs of the apple, where infection results as described for the conidia.

It is to be noted that the conidia may continue to develop in a canker for at least three years. Thus cankers from one

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to three years old are sources of trouble. Furthermore, diseased bark which falls to the ground may develop the ascospores as described above. And finally, the inocula (conidia and ascospores) may come not only from the apple-tree but from the pear, on which fruit-tree the fungus also occurs.

Control.

In applying remedial measures it should be borne in mind that: (1) while the disease appears to be most serious on certain varieties, no variety is wholly immune; (2) the disease is caused by a fungus; (3) the causal fungus infects the trees in the fall, particularly from October to December; (4) the fungus can enter through wounds, lenticels or unbroken surfaces; (5) cankers produced on the limbs are the only source of the inocula — these give rise to all the trouble; (6) these cankers are a dangerous source even when three years old; (7) fallen bark from one-year-old cankers is also a source of the inoculum; (8) the fungus inhabits not only the apple, but also the pear, which may be a source of trouble.

It is therefore out of the question at present to rely on any variety to resist the attacks of the fungus. Any spraying which is done must be accomplished before the most important period of inoculation and infection. It has been shown that old orchards may be cleaned by proper spraying. Bordeaux mixture 6–6–50 is said to be better than lime-sulfur for this purpose. For this disease spray as follows: (1) as soon as possible after the fruit is picked; (2) again within two or three weeks. Where the trouble is serious an application should be made at least once before the fruit is picked; this may be made about September 15, or before the first autumn rains. Then spray twice as directed above. These applications should thoroughly coat the limbs to protect them from the attacks of the fungus. Cankers on the more valuable limbs of younger trees may be removed, in which case the débris should be destroyed and the wound coated with coal-tar. The removal

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and destruction of certain limbs may at times prove helpful. For example, a limb bearing several cankers ought to be so treated. Surgical and pruning measures should in any case be only supplementary to spraying. It should be remembered that the wholesale removal of cankers from large trees where hundreds of lesions occur is an expensive and a laborious method.

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FRUIT-SPOT

Caused by *Phoma pomi* Passer. (= *Cylindrosporium pomi* Brooks)

Nearly all varieties of apples suffer to a certain extent from fruit-spots of one kind or another, but on some varieties like the Tolman Sweet and the Yellow Bellflower there is a specific kind of spotting which is now recognized as an important disease. Other varieties of apples also show this disease, but to a less degree than those mentioned. Sometimes the Baldwin is considerably spotted and thus the disease has been called brown spot of the Baldwin. It has been erroneously referred to as stippen and dry-rot.

In 1892 a fruit-spot was noted on quince in New Jersey. It seems likely that this is the first authoritative record of the

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disease in the United States. Subsequent reports indicate that it is widely distributed throughout the northeastern United States. It apparently does not occur west of Michigan nor south of Virginia. It is said to be common in Canada, and is also reported from Germany. No doubt the greatest losses are incurred by fruit-spot in New England; and often the disease is very appropriately called the New England fruit-spot and the New Hampshire fruit-spot. In these sections of the United States 50 to 90 per cent of the fruit is spotted in epiphytotic years. In the state of Maine the disease is so common on the Yellow Bellflower that the spots have come to be regarded as characteristic markings of this variety, and thus affected fruits have been awarded prizes at fairs and fruit shows. But affected fruit must be graded as a second-rate product, thereby incurring considerable loss to apple-growers concerned.

Symptoms.

The disease has been described on the Baldwin fruit somewhat as follows (Fig. 24): Spots are first seen about the middle of August. They are deeper red on the colored portion of the apple and darker green on the lighter portion. The affected tissue is at first only slightly sunken, if at all. Usually each lesion centers about a lenticel. There are from two to ten times as many spots on the blossom-end as on the stem half of the apple; this is due in a great measure to the larger number of lenticels at the blossom-end. The spots enlarge slowly and never attain great size; the color is finally dark-red, brown or black and the surface of the spot more sunken than at first. In this stage the disease has some semblance to black-rot. At first the spots are superficial, and only in the later stages is the flesh noticeably discolored. In no case does the discoloration extend more than a small fraction of an inch into the pulp. The tissue beneath the discolored skin is rendered brown and corky. Black specks abound on the surface of the lesion: usually one appears in the center of the affected area and others

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are arranged radially about it. These are the fruiting pustules of the causal pathogene. On yellow varieties like the Yellow Bellflower and Tolman Sweet the spots are at first almost a carmine red. Later they turn brown in color. This change in color may occur while the apple still hangs on the tree, particularly if the weather is wet for a few weeks prior to harvesting; or the change may be delayed until the fruit is stored. Spots on affected fruit increase but little in size when placed in cold storage.

Fig. 24. — Apple fruit-spot.

Cause.

The fungus *Phoma pomi* produces this apple fruit-spot disease. The pathogene probably hibernates in the form of sclerotial masses and as peculiar thick-walled cells known as chlamydospores. These structures winter over on fallen fruit. With the advent of the growing season conidia originate from both the sclerotia and the pycnidia on the fallen fruit, and these spores bring about the first infections of the year. Observations show that a larger number of the inoculations take place in July or early August. None occur after the last of August. Entrance into the fruit is gained by way of the stomata. After the spores are deposited on the apple fruit fifty days may elapse before there are any signs of the disease visible to the unaided eye. Owing to this condition of affairs fruits may be inoculated before picking but show no evidence of fruit-spot at the time of harvest. In due time, however, the disease develops in storage or in market. On entering the apple-tissue the germ-

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tube of the fungus rapidly develops a system of mycelium; the threads grow between the cells of the fruit. The large flesh-cells react against the invasion by the fungus as evidenced by their thickened walls. Within the center of such an affected region a small pocket is produced by the collapse of one or two cells. The rate of spreading varies with the variety. After the fungus is well established within the fruit, mycelial masses are developed beneath the epidermis. Ultimately these break through the protective layer and expose themselves as cup-shaped fruiting structures within which conidia are produced. Sometimes pycnidia are developed on affected areas; in these are formed pycnospores which assist in disseminating the fungus.

Control.

This apple fruit-spot is readily controlled by the use of fungicides. Two applications give satisfactory results: the first should be made about the last of June and a subsequent application should be made as late as the middle of July. Earlier and later applications as a rule have little value, while those made early in July are very efficient. If spraying or dusting for apple-scab is practiced, the second and third applications for the scab disease will suffice for fruit-spot. These applications are made (1) when about two-thirds of the petals have fallen and (2) about three weeks later.

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CHAPTER II

APPLE DISEASES — *Concluded*

Soft-Rot or Blue-Mold

Caused by *Penicillium expansum* (Lk.) emend. Thom

Probably no one who has had anything whatever to do with apples is absolutely ignorant of the soft-rot. It is the bane of the apple dealer and consumer, and of any one who attempts to hold this fruit in either common or cold storage. Of all the rots of the barreled apple none is so ruinous, none so common, and none so absolutely destructive. It is world-wide in its geographical range, and it is variously called soft-rot, blue-mold, bin-rot and Penicillium-rot. These names are all significant and perhaps are desirable in the order listed. If the very peculiar and characteristic odor, which is never absent from a diseased fruit, could be accurately described in a single word, doubtless a new and more appropriate common name could be derived. The odor is the first noticeable feature of this fruit decay, but the softness of the affected tissue will never be overlooked by the careful observer, so that the name soft-rot is very desirable.

Symptoms.

The odor given off from the barrel or bin by apples affected with soft-rot has been mentioned; this is a very accurate diagnostic symptom so far as determining the presence of the disease in a lot of fruit is concerned. Apples which do not show soft-rot lesions may, however, carry the peculiar characteristic odor of the disease, owing to contact or proximity with

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affected fruit. The rotten area itself is very soft, watery and light or yellowish brown in color. In the case of lesions involving a considerable portion of the apple the skin becomes wrinkled (Fig. 25), sometimes in a concentric manner. These changes are accompanied by a characteristic moldy taste which is decidedly unpleasant. Young spots may begin anywhere on the surface of the fruit where there is a rupture in the skin. A single lesion may develop at such a rate as to involve the whole fruit in two weeks or less. The rot is primarily one of ripe apples; green fruits are, as a rule, not affected. Under conditions of high relative humidity bluish or greenish blue mold tufts develop profusely over the diseased area (Fig. 25).

FIG. 25. — Soft-rot, or blue-mold.

Cause.

The most important organism concerned in soft-rot is the fungus *Penicillium expansum*. It is very likely, however, that several other species of Penicillium may at times be responsible. The above-mentioned pathogene is generally familiar as an enemy of fruits. It grows as a saprophyte on a large number of dead organic materials and produces a vast number of spores which are omnipresent. These spores float abundantly in the air and ultimately come to rest on various objects, fruits included. Whenever one of these spores falls into a wound of any

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sort on the surface of an apple, it germinates and its mycelium proceeds to feed on the ripe fruit. Among the common types of wounds through which *P. expansum* gains entrance may be noted, finger-nail cuts, bruises, worm-holes, scab spots, and spray-injured places. The fact that the soft-rot fungus gains entrance to the apple through such injuries is highly important in the matter of controlling the disease. It is further worthy of attention in this connection that this pathogene cannot enter normal, unbroken skin. Accordingly it will be learned on observation that in barreled apples of a good quality those fruits at the heads of the barrel are rotted more extensively than those in the center of the barrel.

Like many fungi, *P. expansum* develops an extensive system of mycelium in the lesion. From the germtube, hyphæ grow in all directions into the flesh. The threads dissolve their way between the cells of the fruit-pulp, as a result of which the latter are easily pushed out of position. They slip over each other at the slightest pressure. Thus when the surface of a rotted area is pressed by the finger the tissue quickly and easily gives way, offering no resistance whatever. The disease is well named soft-rot. As already mentioned, under conditions of high relative humidity greenish cushions or pustules appear on the surface of the affected area. These are tufts of fruiting stalks of the fungus which arise from the mycelium within. Numerous hyphæ grow in erect fashion at the same point; their general arrangement is not unlike that of an inverted broom without the handle. The tips of these hyphæ, or conidiophores, become branched in a digitate fashion, and at the end of each stalk is developed a chain of spores, or conidia. The number of conidia which might be produced on the surface of a single rotten apple would aggregate millions. As soon as mature these spores are easily broken from the conidiophore and, being extremely light in weight, float about in the air for some time.

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Control of soft-rot.

It is to be remembered that the spores of the soft-rot fungus are everywhere in the air, and on objects of all sorts. Rotten apples cannot be cured. But obviously there are many things which the grower and dealer can do or can refrain from doing which will reduce the disease to a profitable minimum. These facts should be borne in mind: (1) the fungus is omnipresent; (2) it gains entrance to apples only through wounds; (3) a rotten apple is a menace to its neighbors in the barrel or bin. Therefore, precautionary measures should be used in handling the fruit. Certainly the modern commercial grower expects to raise apples free from scab; at least there are two reasons why he should: to avoid scab itself, and to indirectly prevent storage rots. A scabby apple is not a number one when it goes into the barrel, and no miraculous handling of the fruit can bring it out in better condition than it was in when barreled. The chances are it will be rendered worthless through the agency of the soft-rot organism which finds easy entrance at the scab spot. For most manual labor a careful manicuring is not essential; but for picking apples it is highly important that the finger-nails be short or that smooth gloves be worn. No careful grower will approve of having the fruit bruised in any way during the picking and packing operations. Every precaution should be taken to avoid bruising the tissue or breaking the skin of the apple. Cold storage cannot mend these wounds, nor does it seem to wholly prevent soft-rot. Doubtless cold storage does a great deal toward lessening the amount of soft-rot from year to year. Some refer to the process as refrigeration; but in any case the principle involved is briefly this: the temperature is reduced to a point where the fungus can develop no further, but at which point the apple is not injured. In proper storage the fruit is not only not injured, but benefits, other than by the prevention of decay, are derived. The period for distribution and for consumption is increased. When apples are

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stored in small containers, the fruits are cooled more easily than in larger ones. In a barrel, for example, those fruits in the center remain warm for several days after being placed in cold storage. Thus if wounds of any sort are present on the apples, the fungus gets a good start. Moreover, if apples are allowed to stand in a shed or railroad car for several hours, they become warm; likewise those picked in warm weather and allowed to remain in the orchard will also become warm, so that the temperature in the center of the barrel is reduced but slowly in storage. If the temperature in the center of the barrel is 75° to 80° Fahr. when stored, it will require about one week for the temperature of the air in that part of the barrel to be reduced to a point equal to that of the cold storage room. It has been found that apples held at 32° Fahr. for two months showed soft-rot on removal. At a higher temperature, of course, the fungus develops even more rapidly and the destruction is greater. At an average temperature of 47° Fahr., ranging from 35° to 56° Fahr., the fruits may be three-fourths rotted in five weeks; at an average temperature of 60°, ranging from 48° to 69° Fahr., the whole of each attacked fruit is involved within three weeks. It is important to note that fruit carelessly handled before being stored is likely to become affected with soft-rot, even if stored at the freezing point for sixty days. Where the storage temperature is higher than 32° Fahr. and the duration of storage longer than two months, heavy losses from decay may be expected.

The question of applying sulfur fumes to stored fruit for the destruction of Penicillium spores has been studied. The conidia can be destroyed by using sulfur at the rate of one ounce to each twenty-five cubic feet of space, but when the fumes come in contact with ripe apples their commercial value is greatly lessened. The sulfur dioxid passes through the lenticels, bleaching the fruit.

Spraying has no direct effect on soft-rot; but the practice is indirectly valuable in the prevention of scab and the elimination

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of insects that injure the fruit. If the stored fruit is fancy, and it is desired to keep it for a long time, wrapping each fruit is of decided advantage in preventing decay. It appears after all that the secret of controlling this disease lies in the careful handling of the fruits throughout all operations necessary to marketing and consumption.

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ARMILLARIA ROOT-ROT

Caused by *Armillaria mellea* (Fries) Quelet

This disease is variously known as the shoe-string fungus rot, crown-rot, mushroom root-rot and Armillaria root-rot. In addition to the apple many other trees and shrubs are known to be affected, some more seriously than others. The discussion is presented under Apple, inasmuch as this host is so generally distributed and because the disease is one of considerable importance to the apple-grower in some regions. In certain sections, however, Armillaria root-rot is more prevalent on other plants. In the state of Washington the chief damage is done to prunes. In Oregon, prunes and apples are most severely affected. In these states the blackberry, raspberry, cherry, plum, gooseberry, peach, currant and loganberry are at times badly injured by this root-disease. In Europe the trouble is found on forest trees, including the birch, beech, walnut, oak, chestnut, ash, pine, larch, alder, fir, willow and cotton-wood. These trees are liable to affliction in America, but as already intimated the disease in this country affects chiefly our more

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important fruits. In addition to the long lists of plants already given, the following may be added as minor hosts: olive, grape, crab, maple and potato. It is obvious, then, that a great number of widely related plants are likely to be affected by this disease. This fact complicates the matter of control, and is a factor in the economic importance of the disease.

It is believed that Armillaria root-rot was observed in America as early as 1887 on grape near St. Louis, and later in the states of Texas and California. At present the regions most infested are those of the Central, Southwest and the Pacific Coast region. The disease has attracted particular attention along the Pacific Coast during the last fifteen years.

The damage done by this disease would be difficult to estimate. But bearing in mind the fact that all of the more common fruits are susceptible, and in many localities are severely injured, it is not impossible to gain a fair impression of the amount of losses incurred. In the state of Arkansas it is stated that the losses due to root-rot exceed those of any other disease of the apple. In other localities doubtless a similar condition exists. As has already been pointed out, the disease in some states is more severe on the apple than on other fruits; again, the reverse is true. It may not be out of place to cite an example of the possible damage that this root-rot may do. In a western prune orchard of about one thousand trees, an average of about eighty trees died each year for seven consecutive years as a result of Armillaria root-rot. In seven years half the orchard was destroyed. In another case, an orchard of five thousand trees was reduced at the rate of two hundred and eighty trees per year for three consecutive years, the total trees killed being eight hundred and fifty. Losses of this kind are always keenly felt.

The disease assumes importance in one or more of the following ways: (1) the roots are killed and hence the trees die in one or more seasons; (2) the crown may be injured to such an

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extent that the tree may be blown over under the strain of a heavy crop of fruit; (3) affected trees in bearing usually fail to mature their fruit, particularly in cases of severe infection; or the fruit matures poorly, is stunted and is of an inferior quality; (4) diseased trees often lack the normal amount of foliage; (5) affected plants make little or no growth.

FIG. 26. — Armillaria root-rot; note the girdling of the root.

Symptoms of Armillaria root-rot. Evidence of this trouble varies with the part affected and there are no visible signs until considerable progress has been made by the pathogene. A striking characteristic is the marked localization and the slow development of the disease. The average observer will not suspect the presence of root-rot until individuals or groups of trees die among many other apparently healthy ones. At some point it will then be found upon careful examination that the tree is girdled (Fig. 26) and that finally the top dies. Accompanying this phenomenon is a profuse development of string-like, hard, black, shiny, much-branched strands, $\frac{1}{25}$ to $\frac{1}{12}$ of an inch in diameter, in the soil at the base of the tree (Fig. 27). These strands have a peculiar mushroom odor and a tough texture. From these rhizomorphs, as they are called, numerous white-gilled, honey-colored mushrooms may arise in the autumn (Fig. 27). The mushrooms may be found at the base of the affected tree or at some distance along the roots.

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The first external evidence of Armillaria root-rot is that of a poor growth of the affected tree, accompanied by a yellowing or a wilting of the leaves in midsummer or later. In this stage the tree is beyond recovery, for infection in such a case occurred one to three years previously. Old and young trees are alike attacked by this root-rot pathogene.

Cause.

This root-rot is caused by a mushroom known as *Armillaria mellea*. Sometimes the common names honey mushroom, or honey Agaric, are applied to it, on account of its light yellowish brown color. It occurs widely in the woods, orchards, berry patches and in newly cleared lands, and is extremely variable in form and appearance. The long list of plants which it may attack affords opportunities for an almost certain perpetuation. It may live as a saprophyte on buried wood, spreading through the soil by means of its rhizomorphs. In some fashion or other, the roots of the doomed tree are injured, and through such wounds the rhizomorphs (Fig. 27) of the pathogene enter the bark. Some contend that they enter roots through uninjured bark. The roots may be penetrated by way of the trunk. The rhizomorphs upon entering the root tissues spread out into the separate hyphæ which make up the strand. The hyphæ, or mycelial threads, grow between and into the root-cells, killing them, and finally the root decays. Destruction of the tree

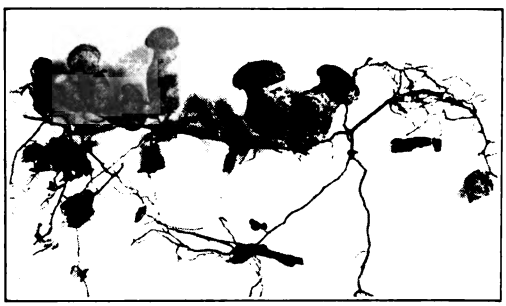

FIG. 27. — Armillaria root-rot fungus; rhizomorphs and young sporophores.

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probably never proceeds more than four inches above the ground on account of a lack of moisture. In living or dead roots and root-stocks the pathogene may live for several years. As already mentioned under Symptoms, the mushroom itself develops from the rhizomorphs in the months of September, October and early November, and rarely in the spring. The mushroom obtains food from the rhizomorphs, and maturity is thus reached in a very short time. At full growth the mushroom varies from three to seven inches in height; the cap measures from two to six inches across; the cap is conical, yellowish at first but becoming dark with age; the gills are white, with reddish brown spots; the stem is swollen near the base, and possesses an annulus which is conspicuous in early stages of development but sometimes only slightly developed or even wanting. The mushrooms often grow in clusters, and, while edible, are not choice.

The system of rhizomorphs which attacks the roots actually travels through the soil from tree to tree. Thus infection occurs underground. From the gills of the mature fruiting bodies, or mushrooms, spores are liberated in great numbers. These spores are carried through the air for long distances and doubtless account for isolated cases of Armillaria root-rot. The spores germinate in the soil, feeding on humus, and produce mycelial threads which unite to form the cord-like strands, or rhizomorphs.

Control.

If trees are once affected, there is little hope of saving or curing them. Nursery inspection is not reliable. The rhizomorphs may be broken off accidentally or intentionally, and thus there would be no certain evidence of the disease. In such a case, too, the tree concerned may have just been infected. In this way a diseased tree does not appear to be infected, and therefore it is certified. Thus the fungus is carried in nursery-stock.

As with a great many plant diseases, the elimination of the

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source of the inoculum offers some relief as a control measure. In selecting a site for orchard-planting care should be taken to determine whether the fungus is in the soil. Newly cleared land should be held in high suspicion. For the pathogene, as already pointed out, may live in the soil as a saprophyte on dead parts such as roots, chunks and stumps. It is a better policy to grow crops other than those listed as susceptible to Armillaria root-rot for a period of at least three years. Likewise if a diseased tree is removed, another should not be planted in its place for three years. The same applies to a tree which is *dead* as a result of the attacks of this fungus. If it is desired to leave the tree in the soil, it may be isolated by digging a trench to prevent spread of the rhizomorphs to neighboring healthy trees. The trench should be dug near the ends of the roots and need not be more than one foot wide and two feet deep. Throw the dirt toward the tree, since such soil may contain rhizomorphs of the pathogene. In Oregon recent work shows that satisfactory results may be obtained in the control of this disease by an "aëration method." By this method affected trees may be recovered. The roots are exposed about the crown of the tree; all diseased roots and bark are removed; the wounds disinfected; and the roots thus left exposed to the sun during the remainder of the summer. Finally all wounds are covered with some good wound-dressing and the roots are covered with soil the following winter. The application of chemicals to the soil offers some promise, although no experimental data are available on which to base reliable recommendations.

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Clitocybe Root-Rot

Caused by *Clitocybe parasitica* Wilcox

The name Clitocybe root-rot is used to distinguish this disease from other similar root troubles. Like the Armillaria root-rot it affects plants other than the apple, although the host-range is less extensive. The peach, cherry and oak are more important among the other trees damaged by the Clitocybe root-rot.

The disease is confined to the southwestern section of the United States. Affected trees have been reported from Oklahoma, Texas, Missouri, Illinois, Indiana, Georgia, Oregon and California. Greatest destruction has perhaps been wrought in the state of Oklahoma; whole apple and peach orchards are said to have been destroyed by this disease within the short period of two years.

Symptoms.

It may be expected that Clitocybe root-rot will be present most commonly in orchards which have been planted on recently cleared timber lands. It is not known on prairie soil. The most characteristic effect of the disease is the exudation of gum from the crown of the tree. The amount of exudate is at times so great as to unite with the soil, forming a gum-cemented soil about the base of the affected tree. This often becomes hardened, forming a sort of cast about the crown and the larger roots. From a distance affected trees may be recognized by either a yellowing or a wilting of the leaves. As in the case of

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Armillaria root-rot, black rhizomorphs are found adhering to the roots; accompanying these is a peculiar mushroom odor. The sporophores, or mushrooms, or the causal pathogene, appear in groups at or near the base of the diseased tree. Their presence, while not constant, affords the most positive evidence of this root-rot.

Cause.

The fungus, *Clitocybe parasitica*, a mushroom, is responsible for this trouble, whence the name of the disease. It was first found and described in Oklahoma about 1900. The rhizomorphic strands mentioned above are found on the surface of the roots. These can be traced to an organic connection with mycelial bands between the cortex and cambium, that is, just beneath the bark. From these latter, subcortical mats of hyphæ, mycelial threads enter the woody tissue by way of the medullary rays. They grow vertically in the wood-ducts; side branches are given off and these enter adjoining cells through pits in walls, destroying the contents of the invaded cell. A gum is formed; this fills the sap-tubes, thus interfering with the ascent of sap.

The parasite propagates itself in two ways: (1) by forming sporophores, or mushrooms, from the rhizomorphs, and (2) by the growth of the rhizomorphs from one tree to another. In the cases where mushrooms are developed, countless spores are formed on the gills on the lower side of the cap of each. These spores at maturity are scattered by the wind. In suitable infection-courts they give rise to mycelium, and subsequently to rhizomorphs and sporophores. The rhizomorphic strands spread from one point to another by growing through the soil. By this means the fungus may travel several feet to other trees. Rhizomorphs have been found in the soil at a depth of about eight inches. The roots or even the crowns of trees are encountered; entrance is gained through healthy or through injured bark. The flat-headed apple-borer is held responsible

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in many cases for producing wounds through which *Clitocybe parasitica* may enter.

Control.

This root-rot disease is subject to the same remedial measures as those prescribed for the Armillaria root-rot. (See page 100.)

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SOOTY-BLOTCH AND FLY-SPECK

Caused by *Leptothyrium pomi* (Mont. and Fr.) Sacc.

Certain varieties of apples show this very familiar disfiguration. The Rhode Island, Peck Pleasant, Rome, Baldwin and Northern Spy are regarded as most susceptible, although in general the disease is most noticeable on light-colored varieties. English growers find that the Newton Wonder is affected more than other varieties. This disease is also found on pears, particularly on the varieties Anjou and Lawrence.

The injury produced is not deep-seated, consequently the disease is not so serious from the standpoint of destructiveness as would first appear. The growth of the affected fruit is not checked, nor does the real quality of the fruit seem to be impaired. But the apple is blemished and thus it may be rendered unmarketable; at all events, fruit affected with sooty-blotch is less salable than clean fruit. While the chief loss lies in the lowering of the market value of the fruit, it should be noted that later ill effects are sometimes shown in the wilting of affected fruit and even in the decay induced by organisms which are always ready to take advantage of a weakened apple. Although no dollar estimates of losses incurred by this disease are at hand, its economic importance cannot be questioned.

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For the past twenty years the prevalence of the trouble has been noticed commonly along the Atlantic seaboard south of the New England states. But the disease occurs elsewhere; it is found in Canada as far north as Quebec, and is well known throughout the eastern, middle and western states. It was unusually common in 1902 in Rhode Island and Connecticut. Again in 1906 an outbreak occurred in these and surrounding states, particularly in New York, Pennsylvania, Maryland, West Virginia and eastern Nebraska. Special notice of it was made in Ohio about 1890 and in the subsequent years 1896 and 1909. The disease was very prevalent in New York in 1915. It was first recorded from the state of Washington in 1907. In 1910, authorities claim, sooty-blotch was first recorded in England.

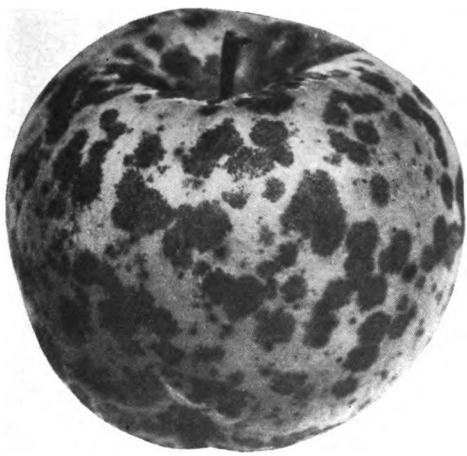

FIG. 28. — Sooty-blotch.

Symptoms.

The names sooty-blotch and fly-speck adequately describe the appearance and effects of this disease. The blotches (Fig. 28) are abundant in the months of July and August. They are irregular in outline, tending to be circular. At first the color is pale, but later, as the name suggests, the color is a sooty-brown or black. On account of the appearance of affected fruit, dealers often call it the cloud or clouded fruit (Fig. 28). Single spots measure from one-fourth to one-half of an inch in diameter; often several lesions coalesce, covering the apple as if with soot. Spots exhibit a radiating structure

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composed of a thin felt of dark-brown interwoven threads which are seen with the naked eye or better with a hand-lens. Fly-speck (Fig. 29), while formerly regarded as distinct from sooty-blotch, is now considered simply as a different symptom of the same disease. The two are found in the same situations and under similar conditions except that fly-speck develops later than sooty-blotch. Fly-speck needs little description (Fig. 29). Groups of a half dozen to one hundred black, shiny dots appear on the surface of an apple; the specks resemble closely those made by the fly (Fig. 29). Both sooty-blotch and fly-speck are very superficial in nature, and there is no hardening or cracking of the apple skin as in scab.

FIG. 29. — Fly-speck.

Cause.

The pathogene causing the disease was described on Newtown Pippins from the state of Pennsylvania in 1831. More attention was paid to the fungus than to its effects on the apple. It is suggested that, since it was discovered in America nearly a century ago, the pathogene was carried across the ocean from the United States to England. It has frequently been observed, for many years, in English markets on American-grown apples. On its discovery in the latter country, in 1910, its history with particular reference to its origin aroused great curiosity. English growers of fruit had not forgotten their experience with in-

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troduced parasites, especially those of such alarming habits as the gooseberry-mildew fungus and others.

The radiating threads which compose the blotches are hyphæ of the fungus *Leptothyrium pomi*. Likewise the black specks belong to this fungus; they are sclerotial bodies. It is unknown just how the causal fungus passes the winter. It has been suggested that it hibernates on the apple-twigs, presumably as specks or sclerotial bodies. In the late spring each sclerotial body undergoes certain developmental changes which result in a pycnidium. Conidia, developed within the pycnidia, serve to bring about the first infections in the summer. On the surface of the apple a conidium germinates in the presence of moisture with the result that a radiating growth of mycelium is initiated. Apparently inoculations do not occur prior to the month of July. The threads extend themselves superficially, or at most penetrating the cuticle but to a slight degree. As the hyphæ grow, they branch uninterruptedly until there is a prostrate, soot-colored mat of fungus threads; these compose the blotches, whence the name sooty-blotch. The cells of the radiating hyphæ become enlarged and function in propagation by breaking away and causing new infections. This probably composes the inoculum during midsummer; for at this season no fruiting structures and no spores are to be found. When the fungus develops the fly-speck stage, the sclerotial bodies may possibly break away from the skin of the apple and may subsequently be washed to new quarters, where secondary infections may occur. This point, however, needs confirmation. In all cases the fungus is highly favored by damp situations and it is most abundant in seasons of considerable late summer rainfall. Little difficulty is experienced in dry seasons; in some years sooty-blotch and fly-speck are practically unknown.

Control.

Fortunately the disease may be kept under control by the methods of treatment employed for other more serious diseases

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like apple-scab. If the schedule advised for the control of scab (see page 12) is rigidly followed, satisfactory results so far as sooty-blotch and fly-speck are concerned will be obtained. Particular attention should be given to the treatments in July and August.

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Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

This disease, known as crown-gall, crown-knot, hairy-root, woolly-knot, woolly-root and root-gall, affects more particularly nursery stock. It is said that the Ben Davis, Early Harvest, Yellow Transparent, Wealthy, Grimes, Northern Spy, Oldenburg, Wolf River, Red June, Gano and Rome Beauty are most susceptible.

Although crown-gall has been known in Europe for many years, it is the belief of certain authorities that the pathogene is native to this country. American nurserymen have known it

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for at least a half century. It is now found on practically every continent of the globe. Records come from Europe, South Africa, Asia, New Zealand and North America. It occurs in practically every state in the Union, but is most abundant in the more southern states of the apple-growing belt, from Virginia to Texas. The disease is also known in Canada.

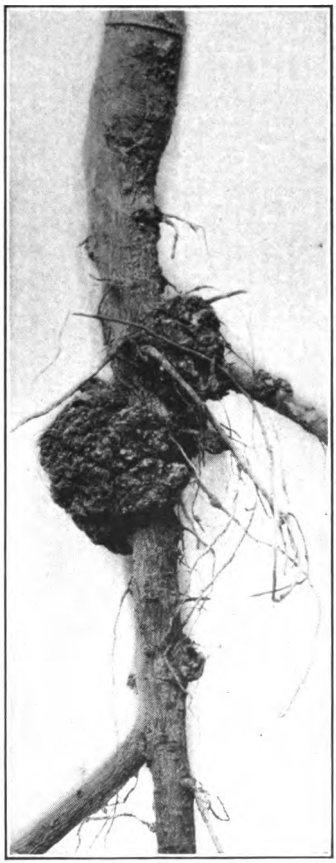

FIG. 30. — Crown-gall on apple roots.

As already intimated, crown-gall is most damaging in the nursery, and becomes most prominent and important where root-grafts are carelessly made. In many instances the affected plant shows no ill effects, and trees are said to outgrow the disease if they are well rooted. In certain other cases affected plants are dwarfed, which of course renders them less valuable. A common source of loss lies in the fact that other pathogenes enter plants through the gall-lesions.

Symptoms.

The disease may exhibit itself in two forms: (1) as galls (Fig. 30), and (2) as hairy-roots (Fig. 31). The galls are either hard or soft and occur at the ground-line or on limbs. It is estimated that fully 90 per cent of the galls appear, in the nursery, on the scion just above the

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point of union with the root. Mature galls may measure from one to several inches in diameter, are dark in color, with a roughened surface, and usually hard. Young galls are comparatively small, greenish, or sometimes slightly flesh-colored, relatively smooth, and soft or even spongy. The second form of the disease, hairy-root, is quite unlike the gall form just described. As the name suggests there is an excessive production of small fibrous roots, which give to the system a hairy appearance (Fig. 31). These may be grouped into several types, as follows: (*a*) a simple type, in which numerous small roots grow out at nearly right angles single or in tufts from an older root or stem; (*b*) a woolly form which originates from a smooth irregular swelling usually on the larger roots near the surface of the soil; (*c*) a broom-like formation in which there is excessive branching of the roots at the ends. Both the crown-gall and hairy-root forms of the disease

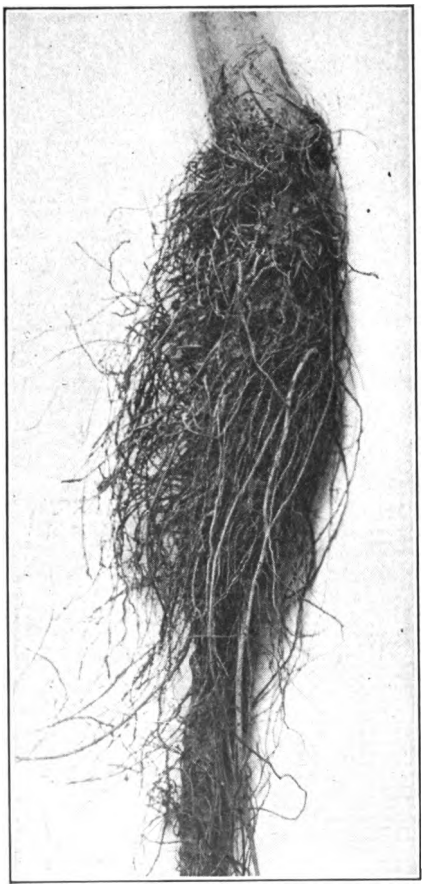

FIG. 31. — Hairy-root of apple.

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prevail in the nursery. The latter form is the more common in orchards.

Cause.

Extensive and reliable experiments have shown that this disease in its widely different forms is bacterial. The organism, *Bacterium tumefaciens*, has been known but a short time, and all points in its life-history are not fully understood.

It is unknown just where the bacteria hibernate, but most likely in the soil. They may live independently scattered through the soil, or in old galls which may persist from year to year. It is thought that the bacteria are carried from tree to tree: (1) by water of irrigation; (2) by cuttings from diseased plants; (3) probably by the pruning knife; and (4) by insects. The bacteria enter the host through wounds, and as a result of their rapid multiplication and stimulative action, the attacked plant shows signs of crown-gall within a week or less. The plant tissue is invaded and the bacteria are found in the cells, but death does not result. On the other hand, the cells are stimulated to excessive multiplication. Soon after the first or primary gall is produced, particularly if the plant is rapidly growing, affected cells push out in strands, into the normal tissue, along the lines of least resistance. This effect is not visible externally, and is seen internally only with the aid of the microscope. The presence of such a strand is evidenced by the fact that the removal of galls is not an effective control measure; new galls arise after the excision. As the strand of diseased cells proceeds, bacteria are carried to points, above and below the primary gall, where secondary galls arise. This manner of originating secondary galls represents a phenomenon similar to that exhibited in the case of malignant animal tumors or cancers; because of this similarity the disease is sometimes referred to as plant-cancer.

It is understood then that infection may arise internally and externally. The bacteria may come not only from the

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apple, but from many other plants, including the almond, apricot, beet, blackberry, cherry, chestnut, clover, cotton, daisy, English walnut, grape, honeysuckle, hop, lettuce, peach, plum, poplar, potato, prune, quince, rose, tobacco, tomato, turnip and willow. This list is incomplete but will give an idea of the wide range of plants susceptible to the disease, and therefore of the many sources from which the bacteria may come to the apple.

Control of crown-gall.

In dealing with crown-gall, the following points should be borne in mind: (1) the great number of plants attacked; (2) the identity of the bacteria causing hairy-root and crown-gall; (3) the greater abundance of the disease in nursery-stock; (4) the bacterial nature of the disease; (5) the entrance of the pathogene into the host through wounds; (6) its presence in the soil; (7) the distribution of the pathogene in nursery-stock; (8) internal infection by tumor strands, and that therefore not all galls necessarily arise from an external source. In the light of these data the following measures and precautions should be employed: (1) deep planting to protect the plant against frost-injury; (2) avoid injuries to the roots and stems; (3) remove diseased trees; gall excision is unsatisfactory and unreliable; (4) protect grafts at the point of union by using a root and scion of approximately the same size, and by wrapping them carefully; (5) reject diseased nursery-stock. (See Table of Contents for other fruits which are affected by crown-gall.)

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Powdery-Mildew

Caused by *Podosphæra leucotricha* (E. and E.) Salm.

This mildew affects, in addition to the apple, the pear, quince, cherry, plum, thorn and juneberry. Seedling nursery-stock, wherever apples are grown, are likely to suffer to some extent from this disease. In localities where the relative humidity at times runs high such varieties as the Yellow Newtown and Yellow Bellflower are liable to be severely injured by this disease. In the Pajaro Valley of California no variety is immune, although there is some variation in this respect. The most susceptible varieties include the Smith, Missouri, Esopus and Gravenstein in addition to the two already mentioned. Those less seriously affected are: White Pearmain, Winter Pearmain, Red Astrachan, Rhode Island and Langford.

Apple powdery-mildew has a wide geographical range over the world. It has been reported from Europe (Germany, Austria-Hungary and Russia), Asia (Japan), Australia, New Zealand and North America (United States and Canada). In the United States it has for many years been a serious nursery pest, and recently the disease has caused injury to bearing orchards. This mildew is most prevalent and most serious west of the Rocky Mountains, particularly along the Pacific Coast where climatic conditions apparently are very favorable to the pathogene. The peculiar weather conditions in the Pajaro Valley of California favor powdery-mildew, and here commercial apple orchards suffer more than those of any other district in the United States. Bearing trees

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elsewhere in California also suffer extensively from the effects of this disease.

The nature of the injury induced by powdery-mildew makes an estimate of the financial loss difficult. The leaves and twigs are directly affected, while the fruit escapes almost entirely. Damage may result to the affected host in one or more of the following ways: (1) the leaves are so affected that it is difficult to find normal foliage in humid regions; this is especially true of susceptible varieties; (2) affected shoots are stunted; the terminals as well as the leaves die; (3) the floral organs are reduced in size; (4) affected trees may fail to form blossom-buds; (5) diseased trees obviously cannot produce well; (6) trees which are affected year after year decline in normal appearance, owing to the cumulative effect of the disease.

Symptoms.

Early in the summer the foliage and young twigs may show signs of powdery-mildew. Leaves are most commonly affected on the lower surface, where a whitish or grayish powder develops. These powdery spots may reach nearly an inch in diameter; several may occur on a leaf, and as a result of their coalescence the lesions involve considerable area. Affected leaves crinkle, become stunted, and are particularly more narrow than normal leaves. Later the mildew may be seen on the upper surface of the foliage. Diseased twigs may be practically covered with mildew. Like the leaves they are stunted, the internodes of a single year's growth sometimes reaching not more than one or two in length. In the winter the terminal shoot may die back; the following spring a new shoot arises from a lateral bud. This phenomenon may be repeated year after year. In the latter part of the summer black fruiting bodies appear as specks scattered through the powdery surface growth. No direct injury is done to the fruit and infections thereon are extremely rare. When the floral parts are affected, they are dwarfed and deformed.

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Cause of powdery-mildew.

The pathogene is a fungus known as *Podosphæra leucotricha* [= *Sphærotheca Mali* (Duby) Burr.]. Sometimes a closely related species, *P. Oxyacanthæ* (DC) DeBary, attacks the apple, causing much the same difficulty as that just described. But in the apple regions of New York and California where the disease has been carefully studied it has been found that *P. leucotricha* is by far the more common form present.

The mildew that is found on the leaves and twigs is made up of interlaced fungus hyphæ. Certain of these threads, or hyphæ, become erect and bear chains of conidia. In mass they give to the affected organ a powdery appearance. These spores, or conidia, are blown to other leaves and twigs throughout the growing season, causing infections. A spore falling on a leaf or twig soon germinates by protruding a germtube. From the latter, mycelium develops copiously, growing out in a radiating fashion. At frequent intervals the mycelium sends haustoria into the epidermal cells of the attacked part; through these organs the parasite obtains food. Soon a new crop of conidia is developed, as already described. During the latter part of the summer dark-brown, globose perithecia are formed among the hyphal threads. These appear to the naked eye as irregular, smoky patches on the twigs. The fungus is carried over winter by these bodies. Each perithecium has several appendages, and a single ascus containing sexual spores. These ascospores are capable of producing infection with the advent of the growing season. It is said that in the Pajaro Valley where powdery-mildew is so prevalent on the apple the ascospores do not commonly bring about first infections in the spring. In that region as well as in others a dormant-bud infection occurs. The lateral and terminal bud-scales are penetrated by the fungus; here it remains dormant until spring, at which time growth is resumed. Hence infected shoots appear as soon as the leaves develop. Conidia mature rapidly on these shoots

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and soon the parasite has extended itself to other twigs and leaves in a manner conducive to certain perpetuation.

Control.

The application of certain fungicides supplemented by the eradication of heavily mildewed twigs are satisfactory remedial measures. But the disease is not easy to control. In those cases where copper sprays are still in use, little benefit may be expected so far as the control of apple powdery-mildew is concerned. The chief difficulties lie in the fact that copper compounds produce leaf and fruit injury, and that the mildew fungus is not as susceptible to bordeaux mixture and similar copper sprays as it is to the sulfur sprays. The sulfur compounds are effective under certain conditions of preparation. Sulfur in some very finely divided state is the most efficient fungicide available for use against this disease. Such fungicides as ground sulfur, sulfur flour and flowers of sulfur are all said to be too coarse to yield satisfactory results. On the other hand, precipitated sulfur is particularly effective against apple powdery-mildew. This may be obtained by the use of lime-sulfur solution, diluted one gallon of the concentrated solution to fifty gallons of water to which is added three pounds of iron-sulfate. By stirring the two together a black, muddy precipitate is formed; this contains sulfur in a finely divided condition, iron-sulfid and calcium-sulfate. It has been demonstrated that this spray is effective against apple-mildew both in New York State and in the Pajaro Valley, California. In the nursery the first application should be made soon after the seedlings have developed the first new shoots. Three or four sprayings should follow at intervals of about two weeks. According to experience in the Pajaro Valley, orchard trees should be sprayed as follows: (1) apply the above fungicide in conjunction with the first spraying for codlin-moth; (2) in conjunction with the second application for codlin-moth; (3) three weeks after the second; (4) three weeks after the third. These recommendations, while

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based on experiments made in the above-named region where the weather conditions are somewhat unusual, should serve as a guide in other localities. Doubtless it will be found desirable to vary the schedule tabulated above to suit the conditions obtaining in those apple districts where it becomes necessary to give attention to the control of apple powdery-mildew.

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Pink-Rot

Caused by *Cephalothecium roseum* (Fries) Cda.

This is a disease which assumed greatest importance when the practice of piling and sweating apples was in vogue. At present this method of handling the fruit is not in use, consequently pink-rot gives markedly less trouble. In seasons of unusual dampness outbreaks have resulted in heavy losses. The years 1882, 1894 and 1902 stand out in the history of the apple industry in western New York because of the epiphytotics of pink-rot. Authorities estimate that thousands of barrels of apples were destroyed in one year in New York State on account of pink-rot. Experiences have been similar in Ohio, Michigan

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and Nebraska. Such varieties as the Rhode Island, Fameuse, Fall Pippin, Pound Sweet, Maiden Blush and Twenty Ounce were damaged seriously by pink-rot under the older methods of orchard practice. The recurrence of such losses, however, should hardly be expected under the present conditions and methods of management. However, the disease still ranks prominently among the minor apple troubles, the fruit being affected on the tree and in storage. The pathogene is fairly common in cellar and commercial cold storage, especially on scabby fruit.

FIG. 32. — Pink-rot following apple-scab.

Symptoms.

The term pink-rot is slightly misleading in that the affected tissue is not pink. The name has arisen from the fact that the conidiophores and conidia of the pathogene are pink in color and stand exposed on the surface of the lesion. Pink-rot very commonly follows apple-scab (Fig. 32). Around the superficial, velvety scab spot the apple-tissue becomes brown, sunken, bitter and rotten. Very early in the progress of the disease, the fruiting stalks of the pathogene become evident, at first white and then pink. These symptoms were observed very commonly in the fall of 1915 on scabby Rhode Islands, in New York both before and after harvest. The decayed areas are circular in outline, and vary in diameter, depending largely upon the size of the scab spot which it surrounds, and upon the weather conditions. The lesion is shallow, the affected tissue firm, corky and dry. Growers

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sometimes call the disease canker, but this name should be avoided.

Cause.

Pink-rot is a fungous trouble, the pathogene of which is *Cephalothecium roseum*. The organism was discovered in Austria about 1836 and is now known all over the world. The fungus lives commonly as a saprophyte on dead and decaying vegetable matter. Its spores may be found floating in the air almost anywhere. Consequently when a wounded or scabbed surface of a ripe or nearly mature apple is exposed, these floating conidia come to rest on such places, germinate, and their germtubes enter the apple-flesh. Scab-lesions furnish the most common point of entrance. The upturned skin at the edge and the cracks at the center of such spots seem to be particularly adapted to penetration by the pink-rot organism. The causal fungus cannot enter through the unbroken skin. Sometimes pears, quinces, grapes and plums are attacked in a manner similar to that of the apple.

Control.

The history of the disease shows that it was most troublesome in the days when apples were piled after being picked. Under such conditions the fungus found excellent conditions for growth and worked great destruction. Where apples are still so handled, the practice should be discontinued. Fruits that are free from scab are freer from pink-rot than scabbed apples. It is almost unnecessary to state, therefore, that thorough and timely spraying or dusting for apple-scab should be given strict attention. Cold storage keeps the fungus in check but does not kill it, as shown by the fact that it grows vigorously when removed from such storage to a warm room. However, it is recommended that apples be stored in a dry, well-ventilated room where the temperature is kept at 32° Fahr. Apples picked, barreled and immediately stored show noticeably less pink-rot than those which suffer some delay between harvest and storage. In

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picking, the fruit should be graded carefully, and suspicious or affected fruits should be discarded.

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Water-Core

Caused by abnormal water relations

Some varieties of apples under certain conditions show a peculiar glassiness in and about the core. The Fall Pippin is said to be badly affected, and the disease has been observed on several other varieties, including the Early Harvest, Yellow Transparent, Pound Sweet, Tompkins King and Rambo. In general, summer varieties suffer most.

Water-core has undoubtedly been present in the apple sections of the world for many years, but authoritative records are not old. It was noted in Maryland in 1908, but is thought to have occurred there several years before. Reports have recently come from other regions of the United States which make it certain that the trouble occurs generally over the country, particularly in arid and semi-arid districts. Apple-growers of Europe, Asia, Africa and Australia are familiar with this peculiar disease in the fruit, and the complaints which come from these continents indicate its troublesome nature.

Symptoms.

Affected fruits are found more particularly on the tops of thrifty trees, and on healthy branches which have less foliage as a result of having been pruned back. Shaded fruits may be

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expected to be affected very little in comparison to those which are exposed to the sun. The diseased portion of the fruit is not easy to detect externally, hence the presence of water-core in an apple may not be suspected. However, the experienced observer is sometimes able to detect diseased fruits by their external appearance. Fruits which should be normally green or yellow will show a blush on one side. Red or dark-colored apples do not exhibit such marked external signs of the trouble. On cutting into a water-cored apple, hard, transparent, watery areas are found in the flesh. Such areas are practically always in close connection with the vascular system. The first evidences of water-core are found here. The bundle shows a water-soaked area about it. The lesion may be near the stem or elsewhere in the flesh, since the bundles are scattered. Occasionally these scattered spots may be small, or there may be extensive watery areas near the surface of the fruit, although none extends up to the skin. Affected apples give off a sweetish, fermented odor, and the taste is not unlike that of frozen fruit. In the later stages, liquid is usually found in the seed cavities and the harder inner membrane of the carpels is cracked and covered with hair-like growths, which eventually assume a brownish aspect.

Cause.

The presence of excess water in the affected tissues gives the described appearance. But the factors which bring about this excess water in certain regions of the fruit are not thoroughly understood. It seems certain that fungi, bacteria and insects are not concerned in bringing about water-core. It is generally accepted that conditions affecting transpiration are the prime factors inducing water-core. Reduced transpiration results usually in excessive sap-pressure, and water is forced into the spaces between the cells of the fruit, giving the affected area a glassy, transparent appearance. There is no one factor which can bring about water-core; two or more factors are necessary, and these must favor excessive sap-pressure and reduced tran-

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spiration. The abnormally high sap-pressure, of course, comes only when there is a continuous flow of sap, and is usually accompanied by lessened transpiration. Observations strongly indicate that the more prominent factors inducing water-core are as follows: — (1) Vigor in trees. Excessive growth, especially in young trees just coming into bearing, is favorable. Such trees do not ordinarily bear a heavy crop, but set only a few, abnormally large apples. It has also been noticed that fruits borne near the tips of branches in trees of any age are susceptible to water-core, provided such trees are growing rapidly. On the other hand, trees making a poor growth rarely show glassy fruits. If vigor or unusual vegetative growth be a factor concerned in water-core, then such factors as promote this condition are, in turn, contributing causal factors. High cultivation should be mentioned in this connection. While excessive cultivation alone cannot be held responsible for this disease, it is nevertheless an important factor. (2) Excessive water-supply. Rain or water of irrigation under certain conditions is highly important. If a superabundance of water comes just before maturity of the fruit, and if this excess be accompanied by extremes of temperature and atmospheric humidity, the disease is very likely to appear. Suppose, for example, following a heavy rain-fall the sun warms the soil to such an extent that considerable water is taken up rapidly by the roots. As long as the sun shines the water will be given off by transpiration; but at night the temperature of the air is lowered markedly while that of the soil is not appreciably changed. Thus the roots continue to send water rapidly to the aërial parts, but under the cool atmospheric conditions transpiration is decidedly checked. As a result, the water accumulates in the fruits and there is developed an abnormally high pressure. Finally the water is forced into the intercellular spaces. Other organs do not show signs of this disease; the fruits suffer on account of their inability to offer resistance to this abnormal pressure. Those fruits which occupy terminal

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portions on the branches suffer most because they are exposed to extremes of temperature. Likewise apples on the south or southwest sides of trees are always most affected. (3) Defoliation or reduced foliage-area. Should a tree be defoliated by some pathogene or other cause shortly before the ripening-period, the fruit is likely to suffer from water-core. The decrease in the foliage-area results in reduction of the evaporating surface, consequently an excess of water is liable to accumulate. Severe pruning may also act detrimentally in this way. The removal of a large number of branches means the reduction in the number of leaves, and therefore checked transpiration. Any injury to the foliage, but which at the same time does not cause defoliation, tends to have the same effect with respect to water-core. (4) Excess tension set up in the cell as a result of a rapid ripening process. In ripening, the starch is converted into sugar; water is absorbed by the sugar and there results a high pressure tending to force water into the intercellular spaces.

Control of water-core.

It seems to be out of the question to wholly prevent water-core, since certain climatological factors are so intimately involved. Some authorities say that it is absent where the soil is well managed throughout the year; that it is absent in soil of a loose texture; and that adequately drained soil seldom produces glassiness in fruit. Further observations along these lines are necessary before positive conclusions can be reached. The conditions already enumerated under which water-core has been observed may offer some suggestions as to remedial measures, although prevention or cure are as yet little short of impossible. Among those recommendations worthy of note are: — (1) In thinning the crop those fruits nearest the tip of the branches should be removed, especially in cases where preference is not otherwise to be shown. (2) Cultivation should proceed normally, and a cover-crop should be planted at least as early as the middle of July to remove excess moisture.

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(3) Since a well-drained orchard may be expected to suffer less than one poorly drained, proper drainage should be provided. (4) Where irrigation is practiced, the water should be supplied in reasonable quantities; particular care is essential if irrigation is practiced at or just prior to the ripening-period. (5) If the climate in the region concerned is peculiarly subject to great range in temperature and relative humidity, the above measures should be more rigidly adhered to than where the climate is quite the opposite. (6) The orchard should be sprayed to keep the foliage in good condition; this practice has value in the control of water-core in that the leaves, which are the organs of transpiration, are better able to function. (7) Pruning should not be done just before the fruit ripens, for such an operation lessens the amount of foliage, thus favoring the development of water-core. (8) Fruit should be picked in proper season; it should not be allowed to hang on the tree after it is ready to pick, since this tends to increase the severity of the disease. (9) As soon as the fruit is picked it should go into storage. Under proper conditions of storage affected fruit, unless seriously injured, will entirely recover. The storage should be cool (not cold storage), and of even temperature.

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European Canker

Caused by *Nectria galligena* Bres.

This disease, troublesome for many years in Europe, was noted on apples in America about 1899. At this time it was found doing damage in Nova Scotia and in New York State. From time to time reports of the European canker on apple have come from various parts of United States and Canada, and now it is known to prevail over a wide area on this continent. It has gained most prominence, as an apple disease, in eastern Canada, New York, New England and the Pacific Coast. The disease is perhaps of European origin. It has been well-known there for scores of years, because of its destructiveness not only to the apple, but also to many forest trees. Previous to 1880, however, no profound investigations were made abroad; about this time considerable attention was given to it in German writings. Subsequently the disease has received more than usual consideration in Italy, Austria, Switzerland, France, Australia, New Zealand, Holland and England.

In Europe, this canker is the most destructive and most dangerous of all fruit diseases. Thousands of trees are killed in their prime. In many localities certain varieties can no longer be grown, and there are even districts in which apple-culture has become a real problem as a result of the ravages of this disease. Nor are the losses confined to the apple, nor to fruit trees. It will be less difficult to give an impression of the economic importance of this disease if brief reference to the plants affected is made. Among these are, in addition to the apple, the pear, quince, cherry, gooseberry, lime, beech, maple, ash, alder, hazel-nut, linden, plane-tree, oak, hornbeam, ironwood, dogwood and magnolia. In Germany, for example, beech-stands are often ruined by it. And while the greatest damage done to forest trees is in foreign countries, its importance on the apple makes it a serious pest.

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In America, its range and destructiveness have not reached proportions which even approach those in Europe. Yet in certain sections, already referred to, the canker is becoming troublesome; and it seems to be on the increase.

So far as varietal susceptibility is concerned, little information is at hand for American conditions. But German authorities say that growers have always spoken of cankerous varieties. It is held by some that thick-barked varieties are more resistant than others. Again, the matter of whether a variety is susceptible or resistant is said to depend directly upon its susceptibility or resistance to frost-injury. Certain writers say that varieties of apples that grow erect and open, remain free from this canker, whereas others near by suffer severely. In contradiction to these observations and opinions, it is held that all varieties suffer alike, and that so-called resistant sorts, when planted among diseased trees, soon exhibit a susceptibility equal to that of their neighbors. Any variety, it is said, which is exposed is just as liable to infection as any other. It is admitted by some that weak-growing varieties show larger cankers, because of their poor vigor, yet this is in most cases not worthy of practical notice. In Germany, susceptible varieties include the Canada Russet, Rheinischen Bohnapfels, Harbert's Russet, Norman Bitter-Sweet, Gray French Russet, Hubbardston, Red Cardinal, Canada Reinette, Red Fall Calvill, White Winter Calvill, Winter Goldpearmain and others. Those resistant are: Fursten, Schaffelder, Boiken, Gravenstein, Carpentin, Eiser and Purple Red Cousinot. In England, Warner's King is said to be notably less susceptible than other varieties.

Symptoms.

In later stages it is not at all difficult to detect the presence of the European apple-tree canker, nor to distinguish it from other kinds of cankers (Fig. 33). In the younger stages, however, it is likely to be overlooked by the casual observer; and when noticed it is found to be not markedly different in appear-

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ance from young cankers of other kinds. The first intimation of the disease consists of a slight browning of the bark in a more or less circular area. This is quickly followed by an intense darkening of the affected tissue. The surface of the canker sinks, leaving the edges prominently raised. In a short time a crevice is evident along the margin of the healthy and diseased bark. The affected tissues are killed, become dry, and slough off, exposing the wood, and at the margin a callus develops. The enlargement of these cankers is slow; within a year the length of the lesions may not increase more than one-half of an inch. But they increase in size the second year, and even for as many as a score of years after, thus resulting in large cankers.

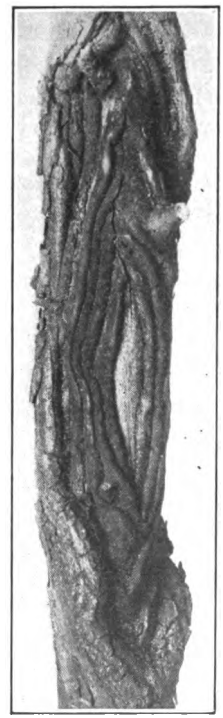

FIG. 33. — European apple canker.

Older cankers are recognized as of two sorts: open cankers and closed cankers; and there are various intervening types. These names are very descriptive. In the first kind, the wound is open, the wood is exposed, and a series of dead callus-layers center about a common point (Fig. 33). While the edge of this type of canker is raised, there is no distinct swelling, and no gall-like appearance. In the closed canker, the whole presents the appearance of a rough gall. The edges are swollen to the extent that the wound is practically closed and the wood almost covered. It is said that open cankers prevail on those erect and more vigorous growing branches and twigs in the crown of the tree. On the other hand, the closed type predominates on the horizontal or hanging limbs. In the case of open cankers the margin

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continues to extend. The prevalence of this type of canker on erect branches is explained on the basis that such limbs lack the necessary food supply to overcome the progress of the pathogene. Here the pathogene appears to have the advantage as evidenced by the expansion of the lesion and the failure of the host to check the spread. In the closed cankers the host has the advantage. Along the margins of the older cankers, brilliant red fruiting bodies of the pathogene develop. These are easily seen with the naked eye, even though they are never larger than a pin-head.

The lesions center about a wound, a bud, or the fork of two branches. Such wounds as those caused by hail, insects, pruning and frost are common seats of the injury. Very commonly a stub or twig may be found at the center of the canker.

Cause.

The European canker is caused by the fungus *Nectria galligena*. As previously intimated the mycelium lives over from year to year in the diseased bark. In the spring and early summer red perithecia develop in the wound and under favorable conditions discharge their ascospores. Conidial tufts are developed at this time of year also, so that there are two kinds of spores for initiating primary infections. It has been shown that insects are highly important as agents of inoculation; the woolly aphis, for example, is very active in carrying the spores of the fungus. In Europe an outbreak of canker is said to follow closely an unusual prevalence of this insect. It has been estimated that in a single canker 300,000 ascospores are available for dissemination. The spores germinate in a few hours and the germtubes enter the bark through wounds or lenticels. Within a week the effects of the fungus are visible. The mycelium, developing from the germtube, permeates the bark, the wood and the pith. The attacks are confined chiefly, however, to the bark, where the cortical cells are killed by the fungus. As a result of death, the affected portion of the bark

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turns brown, the cells collapse, and the canker shows a sunken surface. If the atmosphere is continuously humid, conidial tufts arise from the mycelium. From these tufts, conidia are liberated; they are then carried to other points where new cankers are formed. The mycelium grows more rapidly parallel to the long axis of the limb and hence the canker is the longer in this direction. Where the wood is entered, the mycelium invades the sap-tubes in which it passes up and down. It is believed that at points above and below the canker the fungus again attacks the cortex, this time from within, thus forming a new canker without direct external inoculation. About a year after the canker starts to develop the mycelium forms the red perithecia. These may act as a means of carrying the fungus through the winter.

Control.

The chief measures to be used against the European canker are those of eradication as outlined for the black-rot canker (page 52). The smaller limbs and the badly diseased larger branches should be wholly removed. Smaller cankers on large valuable limbs and on trunks should be cut out, the upper and lower ends of the wound pointed, and finally a wound-dressing, preferably coal-tar, should be applied.

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Superficial Bark-Canker

Caused by *Myxosporium corticolum* Edgerton

In certain of the northeastern states apples and pears are affected with a superficial bark-canker. In practically every apple or pear orchard of New York and neighboring states there is more or less of this disease. But even in those orchards where every tree is affected there is no evidence that serious damage is being done. Every branch of a tree may be extensively affected, while large, bearing limbs commonly exhibit cankers of considerable extent, but in spite of these facts the disease is probably never injurious. Its superficial nature accounts for this in a satisfactory manner.

Symptoms.

The common occurrence of this disease is sufficient in itself to warrant a description. Many growers and even scientists have confused the superficial bark-canker with the black-rot canker. The former disease, like the latter, is found chiefly on the older and larger limbs. But there is no striking depression developed in the case of the superficial canker as with the black-rot canker. At most there is but a slight sinking of the affected bark. The outer bark is killed, and a sharp crevice marks the extent of the lesion (Fig. 34). This line of demarcation is prominent and takes an irregular course on the affected limb. The originally infected areas are small and more or less circular, but large cankers of various shapes finally appear as a result of the coalescence of two or more cankers. Accompanying the pathological changes in the normal bark minute pustules develop in the affected area; these dot the surface and resemble very much those on the black-rot canker. (Compare Figs. 13 and 34.) If

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critical notice be taken, however, it will be seen that the pustules on the superficial bark-canker are of an open, saucer-shaped type, while on the black-rot canker they are closed, flask-shaped bodies. The dead bark clings tightly to the limb for some time. Later, bits of bark fall from the tree. Old cankers usually show considerable checking of the bark (Fig. 34); these crevices are short and sometimes extend at right angles to the long axis of the affected limb.

FIG. 34. — Superficial bark-canker.

Cause.

The fungus *Myxosporium corticolum* is responsible for the superficial bark-canker of the apple and the pear. The known facts connected with its life-history and habits are few. The mycelium of the fungus grows in the outermost bark-tissues only, never reaching the cambium. Scarcely before it penetrates to an appreciable depth its progress is halted by a cork-layer developed by the tree in response to the stimulation induced by the invader. This plate of cork is apparently never penetrated by the fungus, therefore its attacks are confined to the surface cells. The affected tissue is killed and eventually it sloughs away. In this process the cork-layer marks the line of cleavage. During its course of development the fungus forms fruiting bodies, acervuli, just beneath the surface of the bark. At maturity these break through the bark and expose a saucer-shaped interior. Conidia arise from the inner wall of this cavity; and in the moist weather of spring the spores ooze forth in white masses. These spores

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bring about new infections during the growing-season. In the winter the fungus lives in the cankered bark.

Control.

The damage caused by *Myxosporium corticolum* is so slight that little attention need be given to its control. Frequent inquiries are made regarding its nature and treatment, but it is very doubtful whether any sort of remedial measures are ever necessary or profitable.

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SPONGY DRY-ROT

Caused by *Volutella fructi* Stevens and Hall

This disease was reported for the first time from North Carolina in 1907. Since that date it has been observed and recorded from several other states. It is common in New York on apples, but so far as known no other fruit is affected. There are no careful observations on record with respect to differences in the susceptibility of varieties. The disease is known as the spongy dry-rot and the Volutella-rot. The former name is usually given preference.

Symptoms.

The affected area may involve a large portion of the fruit, although the lesions begin as small specks scattered over the surface (Fig. 35). Frequently rotten areas enlarge toward each other, finally fusing into large spots (Fig. 35, right). The younger portion of a diseased area is brown, but the older central portion is coal black. The surface of an affected area is dotted

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with fruiting pustules (Fig. 35, left); these are largest in the central region of the spot and are evident within a quarter of an inch of the margin. If examined with a hand-lens, stiff dark-brown hairs may be observed protruding from the fruiting bodies.

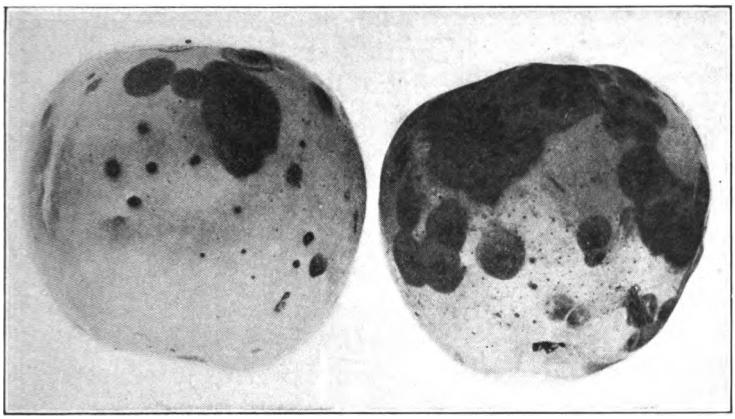

FIG. 35. — Spongy dry-rot; various stages of development.

These constitute an important diagnostic sign of the disease. The affected tissue is spongy and dry, whence the name spongy dry-rot.

Cause.

This apple-rot is caused by the fungus *Volutella fructi*. Within the fruiting bodies mentioned above conidia are produced. These germinate in a manner similar to the process exhibited by the spores of other fungi. The germtubes enter the apple-fruit through injuries of various kinds. Within the flesh mycelium develops profusely and after a few days the rot is evident. Before a lesion has attained a very great diameter the fruiting bodies appear. Mycelium develops abundantly beneath the cuticle and soon a mycelial cushion is formed. This continues its growth until the skin is ruptured and the structure

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protrudes. Certain hyphæ grow erect and parallel to each other; these are the conidiophores. On the tip of each stalk a conidium is borne. From a single fruiting body several hundred spores may be produced. These are scattered to other fruits and thus new infections arise. Intermingled with the conidiophores are dark-brown, hair-like spines called setæ. So far as known the setæ play no part in the propagation of the fungus. They serve, however, to distinguish this pathogene on the apple. It is as yet unknown where and in what condition the pathogene passes the winter. The assumption is, that it lives vegetatively in fallen fruits and possibly on decaying débris of various kinds. Infection does not occur, apparently, before the late summer. Lesions develop most commonly on fallen and stored fruits. The disease seems to be highly favored by conditions that prevail in an uncultivated orchard where the weeds and grass abound; here plenty of moisture is available to the parasite.

Control.

In the absence of experimental data little can be recommended for the control of the spongy dry-rot. The disease is not widely destructive and in even those regions where it is most serious it is doubtful whether special treatment is necessary to satisfactory control. Fruit in orchards that are given the standard care may be expected to suffer but little from this disease.

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Hypochnus Leaf-Blight

Caused by *Hypochnus ochroleuca* Noack

It is in the humid regions of the Appalachian Mountain valleys that Hypochnus leaf-blight is most destructive. The disease is known, however, from Maine to Florida, and is most common in North Carolina, Florida, Alabama, Georgia and West Virginia. The pathogene was probably introduced from Brazil, and is known to attack the apple, pear, quince, snowball and lilac.

Symptoms.

The chief damage is wrought through the loss of leaves. Affected trees are devitalized, and while twigs are killed, this is an indirect injury through the effects of the disease on the foliage. The disease is, then, principally a leaf-blight. From a distance the symptoms are similar in appearance to those of fire-blight, but Hypochnus leaf-blight is clearly distinguished as follows: (1) affected leaves droop and are matted together, a character not exhibited by fire-blight; (2) small sclerotia are present on twigs adjacent to diseased foliage (Fig. 36); these sclerotia are white when young, but are cinnamon-brown in color when mature; they are roundish or oblong and measure one-eighth of an inch or less in diameter; (3) accompanying the sclerotia are found

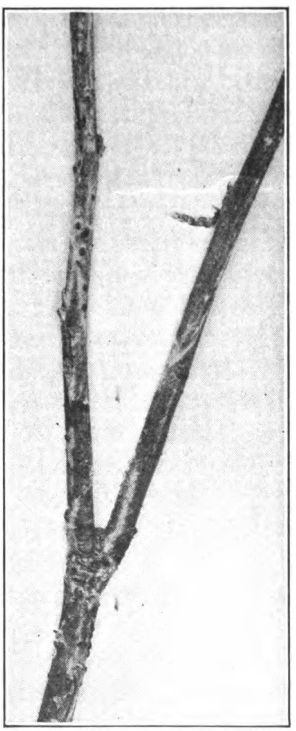

Fig. 36. — Hypochnus leaf-blight pathogene; sclerotia on apple-twig.

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rhizomorphic strands extending lengthwise of the twigs and petioles; (4) in later stages the leaves fall.

Cause.

The causal fungus, *Hypochnus ochroleuca*, hibernates as sclerotia on or near the terminal buds. As new twigs develop in the spring the fungus renews its activities by sending out rhizomorphs from the sclerotia. By this means the pathogene spreads over the lower surface of the leaves, causing them to droop and die, and finally to fall prematurely. In some cases the fruit is an object of attack. The rhizomorphs follow the petioles and main veins of the leaves, finally separating into single mycelial threads. Sometimes mycelial wefts are found on the lower sides of the leaves; these are always connected with the strands or rhizomorphs on the petioles and the strands in turn are in organic connection with the sclerotia on the twigs. Occasionally the weft of mycelium on the lower surface of the leaves assumes a more even texture and ultimately becomes the fruiting layer of the fungus. Here the weft consists of a very closely woven mass of hyphæ. Erect hyphal threads become basidia, each with four spores. The spores are capable of reproducing the structures of the pathogene: the mycelium, sclerotia, rhizomorphs, and finally the basidia. This fruiting stage, however, is rare, and the dissemination of the fungus is efficiently accomplished by the sclerotia and rhizomorphic strands. Throughout the summer the mycelium spreads from one leaf to another, fastening them together in a mat. This matting of leaves constitutes one of the peculiar and striking symptoms of Hypochnus leaf-blight. By midsummer the rhizomorphs form new sclerotia on or near the uppermost buds, where the winter is again passed. The rule seems to be that sclerotia develop in greatest abundance on the distal buds of the twigs where they can be of maximum use in perpetuating the fungus. Sclerotia occasionally develop on the fruit, but in general they are more prevalent on the lower, shaded side of the foliage.

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Control.

Spraying to kill the sclerotia is the only known remedial measure. Special fungicides and special applications are not regarded as necessary. Bordeaux mixture applied before the buds open, and again before the blossoms open, is recommended.

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JONATHAN-SPOT

Cause not definitely determined

A number of different varieties of apples commonly show this peculiar spotting of the fruit. The Jonathan, Esopus, Wealthy, Ortley, Wolf River and some other varieties are severely affected; the disease has been noted on several other kinds, including the Nero, Smokehouse, Newtown Pippin, Yellow Newtown, Grimes, Arkansas Black, Peter, Hibernal and Patten. The common occurrence of this disease on the Jonathan has given rise to the name Jonathan-spot. In Minnesota the growers call what appears to be this disease the Wealthy-spot. Apple orchardists generally hold to the opinion that thin-skinned varieties are most susceptible.

There is no evidence that the Jonathan-spot occurs outside of the United States. Attention was called to the disease in Virginia and West Virginia in 1911. It has been under observation in New Jersey since 1912; in that state it is a very troublesome storage disease. The growers of the Jonathan and Esopus varieties in Washington and Oregon have been the heaviest losers on account of this fruit-spot. It develops both in storage

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and en route to eastern markets. Fruits leaving the Pacific Coast in good condition may show objectionable spotting when placed on sale in the East. This peculiarity of the disease has impaired the commercial standing of the Jonathan to no small degree.

Symptoms.

The spots may appear while the fruit is still on the tree, or after it has been picked. In the latter case they may develop in transportation or in storage. The spots are frequently in great abundance. A characteristic lesion may be described as follows: rarely more than one-eighth to three-fourths of an inch in diameter, it is circular in shape; the color is light-brown at first and with some varieties it remains so for a long time, in consequence of which the spot is inconspicuous. On red varieties the color darkens very early in the development of the lesion. The surface of the affected area is abruptly but slightly depressed. The affected tissue is dry and often only the skin appears to be discolored. In contrast to other fruit-spots it is more superficial than bitter pit, New England fruit-spot, and young bitter-rot lesions.

Cause.

The cause of Jonathan-spot is not certainly known. Several theories and opinions are held in explanation of the causal nature. Some believe that the disease is non-parasitic; others are of the opinion that a species of fungus, an Alternaria, is responsible; and still others champion the opinion that gas causes the trouble. It may be safely stated that none of these ideas and theories are based on conclusive proof. It is very possible that two kinds of spots may occur together on the same fruit; one of which is non-parasitic in nature, and the other caused by some fungus, possibly the one already mentioned. Furthermore, gases such as formaldehyde, sulfur and ammonia may produce spots which are at present indistinguishable from those of fungous or non-parasitic origin. Ammonia from the cooling

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apparatus has been suggested as a causal factor. It has been acceptably demonstrated that arsenate of lead is not the cause of the Jonathan-spot. Spots are found on unsprayed as well as on sprayed fruits. While the cause is yet somewhat obscure it is known that the spotting is more common subsequent to a dry season. The disease was severe following the dry summers of 1910, 1911 and 1914; while after the relatively wet season of 1912 it was less serious. In storage the disease is found most commonly where the fruit is kept in ordinary storage for some time before being placed in cold storage. It also is favored by poor storage ventilation and by improper storage temperatures. Where a relatively high temperature prevails, spots develop abundantly. Under such conditions the fruit often "sweats" and rapidly respires. Apparently the causal factor comes from an external source and gains entrance through the lenticels and through other small fruit cracks as evidenced by the fact that such points serve as centers of all lesions. Since investigators are not generally agreed as to the causal agent, and since the possibility that two or more diseases are confused under the same name, the cause of Jonathan-spot remains to be further investigated.

Control.

Fruits which hang on the tree too long show the disease more commonly than in cases where they are harvested at the proper time. Fruits picked at maturity, rushed to storage, and consumed within a few days after removal from cold storage will not develop the disease to any serious extent. Susceptible varieties like the Jonathan are likely to be severely spotted if the apples are withheld from cold storage, or are merely placed in common storage. It has been estimated that the trouble is quadrupled under conditions of cellar storage as opposed to the amount developed in cold storage. In cold storage the spots which do appear are smaller and less conspicuous. It is to be noted, of course, that the cold storage should be properly ventilated and the temperature should be standard (32° Fahr.).

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AMERICAN BROWN-ROT

Caused by *Sclerotinia cinerea* (Bon.) Schröt.

Apple brown-rot is at times erroneously called black-rot; likewise black-rot is often referred to as brown-rot. The two diseases should not be confused. Apple brown-rot is much less common than black-rot in America, while in Europe brown-rot is by far the more common. In fact, brown-rot is one of the most serious of apple-rots in Europe. In the United States the disease occurs to some extent in several different states, including North Carolina, West Virginia, Virginia, New York, Minnesota, Arkansas, Nebraska, New Mexico and Missouri. It probably occurs in other apple states.

Varieties do not all suffer alike. The disease is more common on summer varieties like the Yellow Transparent and Chenango. In Kentucky, the Genet is injured more than other varieties. In England, a similar disease affects the limbs, forming cankers.

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Symptoms.

The first indication of the disease may appear while the fruit still hangs on the tree, and the symptoms consist in the development of a smooth, brownish discoloration in the skin. The rotted area increases in size, the general form being retained. The pathogene often comes to the surface and shows itself as grayish tufts (Fig. 37); these may be arranged in concentric circles. The rotting of the fruit is finally complete (Fig. 37). In many cases the affected fruit becomes jet black and the skin assumes an ebony aspect. Fruits showing such symptoms usually exhibit no external signs of fruiting bodies of the causal pathogene. The conditions that determine whether an apple affected with the brown-rot disease will remain brown or will become black, as already described, are not well understood. The appearance of these various characters has been explained in relation to weather conditions as follows: — (*a*) if the weather is warm, and the atmosphere has a high relative humidity, the affected fruits become brown, and grayish tufts make an early appearance; (*b*) if the relative humidity is reduced, the fruit is at first brown, then black, and the tufts are rare or absent; (*c*) if the air is dry and cool, the affected fruit is black and no grayish tufts develop. In regions where dry air prevails, this type of the brown-rot disease is the more common. Likewise

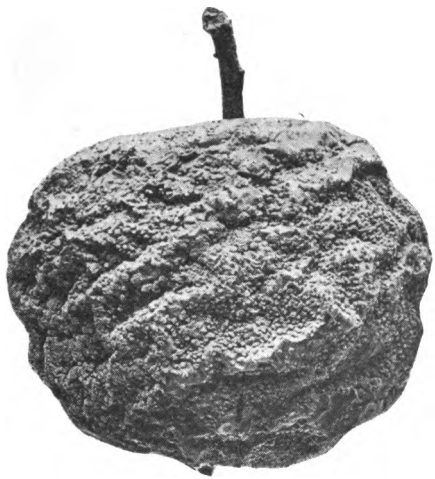

FIG. 37. — Brown-rot on apple.

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under conditions of storage approximating those last enumerated, the brown-rot mummies are shiny and jet black. In any case the interior of the affected fruit is brown and soft. It has been stated elsewhere (page 140) that brown-rot and black-rot are sometimes confused. The following external characters serve to distinguish the two diseases: — (1) Apples affected with black-rot are at first brown, and sometimes remain so, but more often become black, and the surface is dotted with minute black pustules. These characters are sufficient to distinguish brown-rot from black-rot. (2) Apples affected with black-rot shrivel rapidly, become greatly reduced in size and are considerably wrinkled. Apples affected with brown-rot do not shrivel greatly, are not appreciably reduced in size, and are much less wrinkled. (Figs. 10, 11 and 37.)

Cause of brown-rot.

The apple brown-rot pathogene, *Sclerotinia cinerea*, is a fungus which has been confused with *Sclerotinia fructigena*, the organism causing brown-rot of pome-fruits in Europe. Both species may attack both stone- and pome-fruits, but *S. fructigena* invades chiefly pome-fruits in Europe, while *S. cinerea* is the more common form in America. It is doubted that the European *S. fructigena* occurs in this country. The habits and structures of the two organisms are very similar.

Hibernation occurs chiefly as mycelium in the hanging mummies. In the spring conidia are liberated from tufts developed from the over-wintering mycelium. As the fruits come to maturity the conidia, falling into wounds on the fruit, germinate, and finally cause brown-rot. In the autumn many affected fruits fall to the ground; others hang on the tree over winter. It is the latter class which plays the important rôle in carrying the fungus from fall to spring. The history of the fallen mummies is not known; whether a sexual stage ultimately develops from them has not been definitely shown. It is

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reasonable to assume, however, that such is the case, judging from the habits of the fungus on stone-fruits.

Control.

Careful remedial measures have not been determined for American conditions. The suggestion is made on good authority that spraying for apple-scab will help to control the apple brown-rot. Store the fruits in a dry, well-ventilated, and clean house at the customary low temperatures.

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EUROPEAN BROWN-ROT

Caused by *Sclerotinia fructigena* (Pers.) Schröt.

This disease probably does not occur in America, but is discussed in order that a comparison of brown-rot of pome-fruits in Europe and America may be made.

European brown-rot of apple affects the fruits in a manner similar to the American brown-rot (see Fig. 37). But European brown-rot also affects the flowers, shoots and foliage. Diseased flowers are blighted. The woody parts, twigs and limbs, are cankered (Fig. 38). The formation of a European brown-rot canker on apple ordinarily proceeds as follows: a hanging mummy presses against a fruit-spur and the two adhere firmly; the pathogene, *Sclerotinia fructigena*, then grows from the apple-mummy to the branch. An affected branch may be girdled and under conditions of high relative humidity grayish tufts — conidial structures of the pathogene — develop on the surface of the canker. Another method by which the canker may originate is by the passage of the pathogene from an affected blossom through the fruit-spur into the twig.

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FIG. 38. — Brown-rot canker of apple, caused by *Sclerotinia fructigena* (specimen from England).

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ROUGH-BARK

Caused by *Phomopsis Mali* Roberts

This disease is known to growers as rough-bark. It attracted more than usual attention in Virginia in 1909. The more careful observers have noted the disease on the Yellow Newtown; the trouble is so common in Virginia on this variety that at times the roughness is regarded as a character, and is used as a mark of identification. While not common or serious on any other varieties, it is known to affect the York and Winesap. It has recently been found on apples and pears in California.

Symptoms.

The rough-bark disease is most serious on the smaller branches of old neglected trees. The one-year-old twigs are very susceptible, while the current year's growth escapes. Sometimes larger branches and even the trunks are affected. The diseased

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bark sinks in definite areas; these spots are dark, cracked, and have ragged margins (Fig. 39). These characters give rise to the name rough-bark. Some of the lesions spread uninterruptedly until the whole branch is involved. Small branches are thus occasionally killed as a result of girdling. Other affected areas are halted and the wounds are occluded. As a result of the above abnormalities the foliage assumes an unhealthy aspect; it becomes pale and cannot function properly. The leaves are never directly affected under natural conditions.

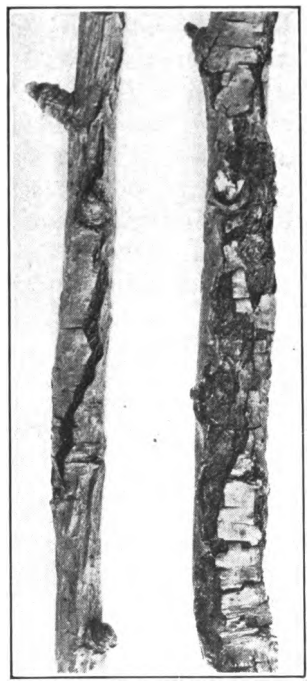

FIG. 39. — Rough-bark canker of apple.

Cause.

Only recently has it been shown that the rough bark of the Yellow Newtown is a disease and not a normal characteristic. The trouble is now known to be due to the fungus *Phomopsis Mali*. It gains entrance to the bark tissues through wounds in the earlier part of the growing-season. While the fungus can be induced under artificial conditions to develop on the fruits and foliage of the apple, yet the lesions on these organs are not regarded as economic phases of the disease. Shortly after the fungus attacks the bark, pycnidia develop from the mycelium. From these fruiting pustules spores ooze forth in cream-colored tendrils. A moisture-period of some duration is essential to this process. These spores are capable of initiating new lesions on the bark. The fungus hibernates in cankers which have been

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developed throughout the growing-season. In the spring the pathogene renews activities, thus perpetuating itself from year to year.

Control.

Spraying is said to offer little hope for the control of rough-bark. In most cases it would be a tedious operation to attempt eradication of all diseased twigs. On the other hand, the decision in this regard must rest with the grower who is especially desirous of cultivating the Yellow Newtown in those apple regions where the disease is most prevalent. The susceptibility of the above-named variety should be borne in mind in contemplation of orchard planting; the experience of the local growers of this variety should be the guide in determining whether it can be profitably grown in spite of rough-bark.

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OZONIUM ROOT-ROT

Caused by *Ozonium omnivorum* Shear

The apple and pear particularly are likely to suffer from this root-trouble occurring in the South and Southwest. To growers in these regions it is probably best known as a cotton disease; however, it affects not only fruit-trees and cotton but also forest-trees, vegetables, forage crops and weeds. The range of host-plants is practically unlimited, a matter of no mean consideration from the standpoint of control.

Symptoms.

Plants affected by this disease usually show threads of the causal fungus on the roots. These threads are at first whitish, then dirty-white or brown. If cotton shows the disease, then

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apples and other trees in close proximity are likely to become afflicted. The disease is recognized on cotton by a sudden wilting, usually during the latter part of June or early July, such symptoms being exhibited by isolated plants here and there in the field.

Apples affected with Ozonium root-rot show a sudden wilting and death of the leaves; this is particularly characteristic of young trees. In case of old trees death is more gradual. In general affected trees have an abnormal, sickly appearance for a year or more prior to actual death. The causal fungus surrounds the tap root as well as the lateral roots; such roots die, decay, and thus cannot function, either in lending mechanical support to the tree or in furnishing the top with water and food.

Cause.

Numerous theories have been advanced by growers to explain the causal relationships of the Ozonium root-rot. But, as already intimated, the fungus *Ozonium omnivorum* creates the disturbance. It lives and spreads in the soil and seems to have a decided preference for the Houston clay or black waxy soils of the Southwest. The fungus grows best where soil aëration is the poorest. A high temperature and plenty of moisture are favorable to its development. Therefore the hot weather of the South favors it; and it flourishes where there is excessive water of irrigation, if other conditions are favorable.

Once in the soil the fungus is washed about, and finally a root is encountered. The bark and woody tissues are penetrated, their cells being killed as the mycelium proceeds; ultimately decay is the result. After its work is complete, the mycelium breaks up into segments which are washed away to other roots of the apple or other plants. It may also spread by growing through the soil on decaying material, or may be carried by tools used in cultivation. No special fruiting bodies are known.

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Control.

To the grower who contemplates planting an orchard in the Southwest it is advised that the list of possible host-plants be carefully consulted before proceeding. If it is determined that the pathogene is present in the soil of the site proposed for the orchard, it is best to wait from three to five years before planting. Select trees from a nursery known to be clean. Orchard cultivation gives proper aëration for the roots and at the same time presents conditions unfavorable to the fungus. Deep fall plowing has been advised for cotton; this measure may assist not only the cotton-grower but also his neighbor who may wish to grow apples. In severe cases the fungus may be isolated by digging trenches about infected trees, going as deep as the roots penetrate into the soil. All weeds, most particularly the sida, should be destroyed. A tree once found diseased ordinarily cannot be saved; this is due to the fact that there is usually no external indication of the trouble until the whole root-system is practically destroyed. Diseased and dead trees should be removed and destroyed. If a stump is left, it is recommended that the dirt be removed from about the roots, allowing them to dry, then that the whole stump and its root system be burned.

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ROSETTE

Caused by adverse soil conditions

This peculiar condition attracted attention in Colorado apple orchards following the severe winters of 1898 and 1899. In 1901 it was particularly noticeable in several orchards of one locality in that state. What may prove to be the same trouble has subsequently been observed in California and Idaho. Records of apple-rosette have not come from any other sources, so that the disease seems to be limited in its geographical range. This fact alone accounts for the disease being considered as one of the minor troubles of the apple.

Symptoms.

The rosette of apple is said to present an appearance suggestive of peach-rosette. The characteristic feature of the disease is the presence of a tuft or rosette of small leaves at the ends of branches; such branches are otherwise nearly destitute of foliage. Affected limbs and even whole trees die from the effects of rosette. In the spring the lateral buds die, and the terminal one develops a clustered branch on which the leaves are much more dense than they normally should be; whence the term rosette.

Cause.

Apple-rosette is called a soil disease. No parasites of any kind are concerned. Like most diseases of this sort the true cause will probably remain in obscurity for some time to come. Conditions accompanying the disease are somewhat as follows: in an excess of marl and an adverse water-supply the tree sends out few or no fibrous roots. In fact the roots cannot penetrate this type of soil to any extent, consequently the tree has a shallow root-system, and the water-supply becomes inadequate. Accompanying these provoking conditions is winter-injury, which comes about in the following way: the water may be in sufficient quantities in the early part of the season, but by the

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last of June the supply is exhausted. The soil readily dries out and the tree suffers from a lack of moisture; hence its growth stops and the tissues harden. In July water of irrigation is supplied and many trees start into growth again; the result is that such trees possess soft and immature tissues which with the advent of winter are killed or severely injured.

Control.

Our incomplete knowledge of the cause of the disease makes the problem of control difficult, or even impossible. The following recommendations based on the information at hand are offered: (1) avoid planting apple-trees in soil where marl is close to the surface; in this way shallow roots and an inadequate water-supply are avoided; (2) increase the depth of the soil by deep plowing before the orchard is planted, by the addition of straw and by plowing under green manures such as vetch, clover or rye; (3) water of irrigation should be used judiciously. The amount of water should be lessened with the approach of autumn in order that the tissues may properly harden before winter.

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SEPTOBASIDIUM-CANKER

Caused by *Septobasidium pedicillatum* (Schw.) Pat. (= *Thelephora pedicillata* Schw.)

This is not a well-known disease. It was first noticed in America about 1889 in Texas and Alabama. In 1911 it was described from North Carolina, and is now said to occur in West Virginia and Georgia. Outside of the United States it is found in Cuba, Ceylon and New Zealand. In this country at least it should not be expected to prove a serious menace to the

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apple. In addition to the apple it is said to affect the pear, oak, palmetto and tupelo.

The causal fungus, *Septobasidium pedicillatum*, attacks the bark, cambium and wood, causing these parts to turn brown and die. Twigs and trunks are subject to infection. Lesions may ordinarily be found at a dormant bud, or at the base of a fruit spur or twig. The forking of branches also furnishes a point of attack. As a result of the work of the pathogene, spots one-half to five inches long are developed; the width varies with the size of the affected branch. In the later stage the bark becomes sunken, and the edge of the lesion is whitish and uneven.

There is little in the way of experimental data upon which to base reliable suggestions for control. It is believed by some authorities that dormant spraying will aid in reducing the amount of the injury. The removal of diseased parts followed by the application of a wound-dressing, such as coal-tar, is worthy of trial.

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Phytophthora-Rot

Caused by *Phytophthora cactorum* (Lib. and Cohn) Schröt.

This interesting disease was given attention in Europe about ten years ago. In certain foreign countries Phytophthora-rot seems to be quite common and destructive. It is said to occur in Switzerland during rainy seasons on apples and pears, rotting the fruit to a considerable extent. The disease also affects and brings about the death of apple "buds" in the Swiss nurseries. An outbreak is reported from Bohemia in 1910, and the trouble has at last been found in America. In 1915 it was discovered

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on Oldenburg apples while still hanging on the tree at Ithaca, New York, by the junior author. Later it was found in market fruit. So far, it has done no great damage in the United States, partly due, no doubt, to its very limited range. Up to date it has not been reported outside of New York State, although it doubtless occurs, to some extent at least, in several of the apple regions of North America.

FIG. 40. — Phytophthora-rot of apple.

The disease is induced by the fungus *Phytophthora cactorum*. Affected apples were first observed in July on those fruits nearest the ground. Earlier infections were not seen. These facts indicate strongly that the source of the inoculum is the soil. Infections occur up to the time of the ripening of the fruit. A brown-rot is produced which in many respects closely resembles fire-blight lesions on apple fruits (Fig. 40). The affected portion appears water-soaked at first, the margin is not definite, and there are no external evidences of fungus fruiting bodies.

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CHAPTER III

APRICOT DISEASES

THE apricot is, as a rule, a very healthy tree. However, there are a few diseases which affect it to some extent. These troubles are practically identical with those of the peach and other stone-fruits. The more important diseases and injuries discussed on succeeding pages are: brown-rot, frost-injury, scab, Coryneum fruit-spot, rust and black-spot. During recent years most attention has been given to the last three. Doubtless brown-rot is the best-known disease of the apricot.

BROWN-ROT

Caused by *Sclerotinia cinerea* (Bon.) Schröt. (or possibly *S. laxa* Ad. and Ruhl.)

The brown-rot disease so common on peaches and plums is also prevalent on apricots in those regions where this fruit is cultivated. It is regarded as a serious disease of the apricot in Europe, causing greater total damage abroad than in America on account of the more extensive apricot-culture in European countries. In the state of California, however, considerable damage is done to young twigs, which are wilted and killed back, and to fruits on the tree, which are rotted as in the case of peaches. Early apricots are said to suffer most, probably on account of moisture relations rather than because of any varietal peculiarities.

No definite schedule of control has been demonstrated, but it is recommended that self-boiled lime-sulfur be applied (1) just

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as the fruit is setting, and (2) later, depending on the amount of rain. In seasons of brief shower-periods followed by drying weather, spraying is regarded as unnecessary. (Brown-rot is more fully discussed under Peach, page 270.)

Frost-Injury

Caused by the action of low temperatures

Like other fruit-trees, the apricot suffers from the effects of frost. Injury by low temperatures in winter, winter-injury, is perhaps the most destructive of all apricot troubles. Collar-rot, or crown-rot, is particularly common and injurious. (For fuller discussion and additional references see under Apple, page 35.)

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Stewart, F. C., Rolfs, F. M., and Hall, F. H. A fruit-disease survey of western New York in 1900. Apricot diseases. Collar rot. New York (Geneva) Agr. Exp. Sta. Bul. 191: 303–304. 1900.

Scab

Caused by *Cladosporium carpophilum* Thüm.

The scab disease, or freckle as it is often called, occurs on stone-fruits other, than the apricot. In fact it affects the peach very commonly. Black scab spots are produced on the fruits and pale-gray blotches with dark margins are developed on the twigs. The first record of the disease on apricot in California dates back to 1909. While it has attracted some attention in that state, yet it is said to have never been sufficiently troublesome to warrant treatment. It has been found abundantly in Connecticut and Texas. (Fuller discussion under Peach, page 294.)

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Reference on Scab

Heald, F. D. Report of committee on fungus diseases for 1911. Apricot scab or freckle (Cladosporium carpophilum Theum). Texas Agr. Dept. Bul. 22: 159–160. 1911.

Coryneum Fruit-Spot

Caused by *Coryneum Beijerinckii* Oudem.

This disease is best known in California on the peach and is referred to as the California peach-blight, shot-hole and fruit-spot. In that state it was given special attention in the years 1907–1909. It has also been noted in New York. In foreign countries it is not unknown, having been recorded from Australia as early as 1882, and an epiphytotic was reported from Algeria in 1904. Small, reddish spots at first with light centers then becoming dark-green to black, are produced on the fruits (Fig. 41). The foliage is spotted; the affected areas are brown, but soon these fall away, leaving a shot-hole effect in the leaf (Fig. 42). Fruit-buds are sometimes killed as a result

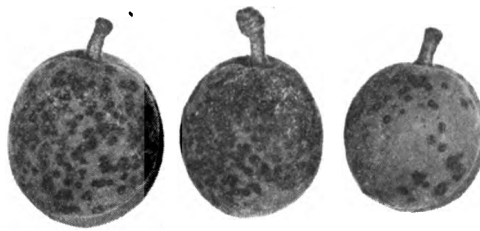

Fig. 41. — Coryneum-blight disease on apricot fruits.

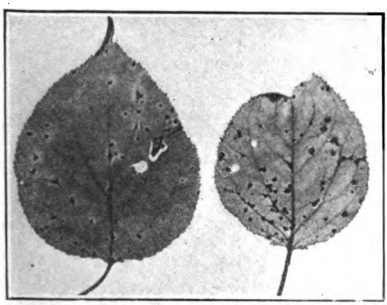

Fig. 42. — Coryneum-blight disease on apricot leaves.

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of an attack prior to blossoming. Recent records indicate that the disease is not easily controlled. (More complete discussion under Peach, page 311.)

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RUST

Caused by *Puccinia Pruni-spinosæ* Pers.

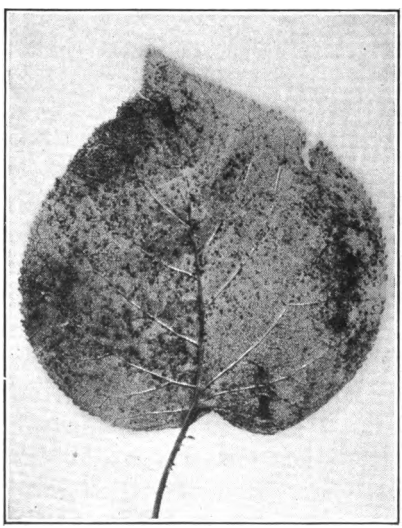

FIG. 43. — Apricot-rust.

This is a disease of stone-fruits in general. On the apricot it is especially prevalent in California. Reddish brown, dusty pustules are produced on the lower surfaces of the leaves (Fig. 43). This occurs late in the summer. The causal fungus, *Puccinia Pruni-spinosæ*, may induce premature defoliation in cases of severe infection. (See full discussion under Plum, page 377. See also Cherry, page 187, and Peach, page 319.)

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Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

In California this disease has been troublesome for many years. Since 1880 it has been the subject of inquiry and writing, with reference particularly to its cause and control. In recent years the question of crown-gall on a great variety of plants has been illuminated, and the more important phases of the disease have been carefully studied. (For a fuller discussion of the disease, see under Apple, page 108.)

Gummosis

Caused by various factors

The formation and exudation of gum is a phenomenon to which the apricot, like other stone-fruit trees, is subject. Gummosis, or gum-flow, is the result of injury due to such factors as mechanical abrasion, unfavorable climatic conditions, bacteria and fungi. (See Cherry, page 181, and Peach, page 303.)

Die-Back

There appear to be at least two distinct die-back diseases, one in California, which is regarded as being due to a lack of water and other factors, and the other has been described from Missouri caused by the fungus *Valsa leucostoma* var. *rubescens*. The latter type also exists in Texas, New York and doubtless elsewhere. The California trouble is evidenced by a dying back of the branches of apricots and almonds. The middle-western and eastern disease shows as cankers on the southwest side of trees. In this connection sun-scald, or late winter-injury, is suggested; and it is not unlikely that temperatures do play a part in bringing about the disease. Large branches die, which is a symptom that may cause some confusion in an

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attempt to distinguish the two die-back diseases. Where *Valsa leucostoma* var. *rubescens* is a factor, its pycnidia show as whitish dots over the surface of the lesion.

(See in this connection Frost-Injury under Apple, page 35, and Die-Back under Peach, page 299.)

Black-Spot

Caused by *Bacterium Pruni* E. F. Smith

As a rule, apricot fruits are less injured by black-spot than those of the other susceptible stone-fruits like the peach and plum. However, certain varieties of apricots are severely affected; the Royal, Sweet Russian, Peach and Bread varieties are prominent in this respect.

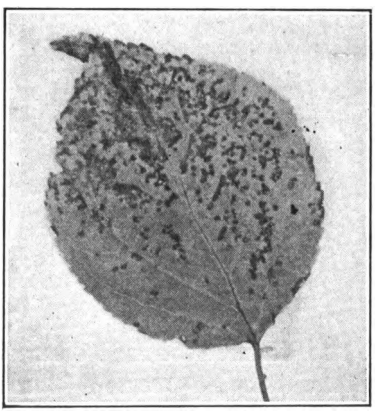

Fig. 44. — Black-spot on apricot leaf.

The disease is also found on the twigs, where it shows as black spots, but these, as in the case of those on nectarine twigs, disappear, and open cankers develop to some extent. On the leaves a shot-hole effect is found subsequent to a dying of small areas (Fig. 44). (See Peach, page 306.)

Silver-Leaf

Caused by *Stereum purpureum* Fries

The apricot is not infrequently affected by silver-leaf. The diseased trees are recognized by the peculiar ashen-gray color

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of their leaves. Accompanying this symptom is the production of little or no fruit. (For full discussion see Plum, page 368.)

YELLOWS

Cause not known

This is a specific disease, showing preference for the peach. It is said to affect the apricot occasionally. (See discussion under Peach, page 283.)

FIRE-BLIGHT

Caused by *Bacillus amylovorus* (Burr.) Trev.

The fire-blight disease of pears, apples and quinces was found on the apricots in Colorado about 1902. It is of relatively little importance, owing to the economic position of the apricot in fruit-culture and also because this fruit is only occasionally affected. Fruits and twigs, when attacked by *Bacillus amylovorus*, present symptoms very similar to those developed on the pomaceous fruits mentioned above. (See Pear, page 323.)

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CHAPTER IV

BLACKBERRY DISEASES

THERE are several troubles with which blackberry-growers have to contend. Crown-gall, anthracnose and orange-rust are the most serious enemies. Leaf-spot is common, but not destructive. The dewberry is severely affected with double-blossom in certain localities. For the most part, blackberry and raspberry diseases are the same.

CROWN-GALL

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

Blackberry represents but one of the many plants attacked by *Bacterium tumefaciens*. The galls (Fig. 45) produced are very much like those on raspberry, except that at the crown they are often larger and frequently occur along the stem above ground. The blackberry suffers to a considerable extent from crown-gall. The tree fruits, like the apple and pear, may at times outgrow the galls, but the bush fruits, such as the blackberry and raspberry, succumb more readily. It seems that the blackberry, like the peach, is more readily infected than some of the other fruits. Possibly this is explained on the grounds that bush fruits are set relatively close to each other, which feature of their culture is more conducive to inoculation and gall-formation.

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(See more complete discussion under Apple, page 108. See also Raspberry, page 398.)

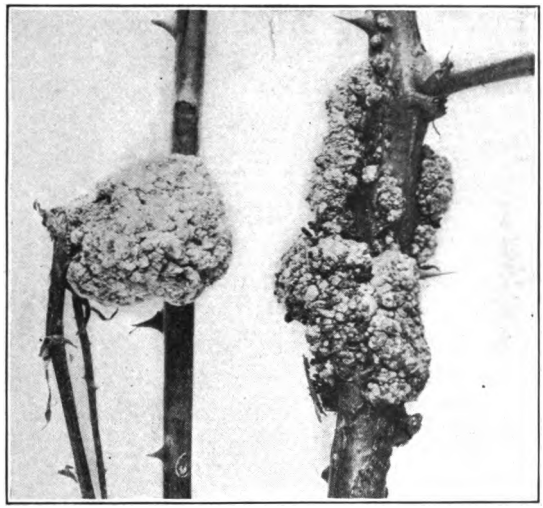

FIG. 45. — Crown-gall disease on blackberry canes.

ANTHRACNOSE

Caused by *Glœosporium venetum* Speg.

The anthracnose of blackberries is the same as that on raspberries. The appearance of the disease on the various susceptible parts is similar in both cases. The causal organism is also the same.

Recent investigations in Washington have shown that blackberry-anthracnose can be profitably controlled by spraying. Two applications are necessary: (1) spray just before the buds open with bordeaux mixture 5–5–50 to which has been added resin-fish-oil soap at the rate of one pound to fifty gallons of the spray. The resin is valuable in rendering the spray more

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adhesive, and the soap will aid in the control of leaf-hoppers. (2) Spray two or three weeks after the petals fall, using burgundy mixture in place of bordeaux. This substitution is made in order that no precipitate will be apparent after the fungicide has dried on the berries; for coating the berries with a spray that shows at picking-time is objectionable. The burgundy mixture is made from the following formula: copper sulfate (blue vitriol) two pounds, sal soda (washing soda) three pounds, and water one hundred gallons. To this mixture is added one pound of soap to each fifty gallons. The soap should be dissolved and added to the mixture after the spray tank is filled. The mixture should be thoroughly agitated during the spraying operation.

Under Washington conditions, profit from this measure of control is obtained through increased quality and quantity of healthy berries, and by the prevention of cane girdling. The practices of cane removal and spraying in the fall have been found to be unnecessary.

(This disease is more fully discussed under Raspberry, page 406.)

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ORANGE-RUST

Caused by *Gymnoconia interstitialis* (Schlecht.) Lagerh.

The leaves of blackberry are commonly affected with orange-rust. The disease appears in the early spring as large, bright, orange-colored patches on the lower surface. The affected leaves are dwarfed and rolled slightly, exposing their rusted lower surfaces. Sometimes the rust patches are found on the canes. As a rule, however, the canes and roots, although often

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infected, show little external indication of disease. It has been observed that affected plants are freer from prickles than normal plants. Healthy and diseased canes may be found in the same stool. All affected canes should be destroyed. The same disease also occurs on raspberries (see fuller discussion, page 399).

DOUBLE-BLOSSOM

Caused by *Fusarium Rubi* Winter

This is primarily a disease of the dewberry, although the cultivated blackberry is affected. The trouble occurs to some extent on the high-bush blackberry. Among the dewberries the Lucretia and Rathbone are notably susceptible, while the Black Diamond and other varieties are less liable to attack.

Double-blossom is most common in the Delaware-Maryland peninsula. It has been observed, however, in Illinois, Tennessee, Texas, New Jersey, North Carolina and Alabama. The first record of the disease comes from Illinois about 1885, although the important writings on the subject of double-blossom are recent.

The history of the disease shows that it does not vary in abundance in a given region from year to year. But, as would be expected, it continues to increase in severity until affected plants are worthless. If diseased plants are allowed to go untreated, their life is shortened by two or more years. In the Delaware-Maryland peninsula double-blossom has been so severe on Lucretias that half of the growers have discontinued dewberry culture.

Symptoms.

Evidences of double-blossom appear early in the spring just as the leaf-buds are opening. The trouble may be detected previous to this by the enlargement of the diseased buds. When affected leaf-buds open there is produced, in place of normal leaves and shoots, a witches'-broom. This abnormal develop-

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ment may consist of short, slender twigs. Sometimes one healthy shoot is found among diseased ones. Shoots of brooms will frequently remain green after old canes are dead. When flower-buds open, they display various malformations: sometimes the deformity is slight, again extensive, depending somewhat on the extent of infection. Blossoms are usually affected, the sepals and petals being thickened. Some growers erroneously regard these diseased blossoms as male flowers. At times the blossoms are increased in numbers; this is especially true of the petals. Diseased petals have the appearance of being doubled, whence the name double-blossom. Other parts of the flower may show abnormal growth, due to the double blossom pathogene. The stamens and ovaries are affected. It happens at times that certain flowers appear healthy, but such flowers may have diseased ovaries. Every bud in an affected plant may be diseased. Sometimes late blossoming is induced in plants attacked by the double-blossom parasite. In such cases the blossoms are smaller than normal.

Cause.

The abnormal bud-development just described is due to the fungus *Fusarium Rubi.* In the early spring its mycelium may be found between the parts of affected buds. With the advent of proper conditions growth is resumed. The mycelium does not pass from the bud into the stem. It does, however, enter certain parts of the bud. The ovaries are penetrated by way of the stigma and the style. An abundant growth of mycelium takes place within these organs, but the carpels and ovules are not penetrated. Neither are the stamens penetrated, although the hyphæ may be found abundantly between them. Wherever the mycelium enters the tissues of a bud, haustoria are sent into the cells. Thus the fungus feeds on its host, stimulating the leaf-buds to witches'-broom formation, and dwarfing the ovaries of the flowers. This influence is also felt by other parts of the flower, as noted under Symptoms.

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Within a few hours after the opening of a flower-bud, the ovaries become more or less whitish, due to the presence of mycelium and spores which develop abundantly thereon. Spores may be produced within forty-eight hours after a flower-bud opens. These are carried chiefly by the wind; it is believed that the clothing of man, tools of various kinds, and insects are also important carriers of the spores.

The spores perchance alight on young buds that are being formed for the following year. In the presence of moisture the spores germinate and produce mycelium which lies dormant in the buds throughout the winter. In the spring growth is again resumed.

Control.

Spraying has not proved to be a satisfactory means of combating this disease. The spores are produced at flowering and during the period of growth of the fruit. Spraying at blossoming might be objectionable, and any spraying done later in the summer must not result in injury to, nor discoloration of, the fruit.

Handpicking deformed buds seems to be satisfactory. The operation should begin as soon as the buds begin to open. As a precautionary measure these buds should be destroyed, preferably by fire. This method is very effective, on account of the fact that the fungus does not go beyond the bud. The grower cannot hope to completely eradicate the double-blossom pathogene, yet the operation is said to be profitable.

In southern regions all canes are cut at the ground immediately after the crop is harvested. This is considered an important operation in dewberry-culture on account of its value in the control of anthracnose and double-blossom.

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Armillaria Root-Rot

Caused by *Armillaria mellea* (Fries) Quel.

The shoe-string fungus attacks the blackberry among other plants. It is said that considerable injury is done in the Pacific Northwest. (See Apple, page 96.)

Leaf-Spot

Caused by *Septoria Rubi* Westd.

The common leaf-spot of blackberry affects also the raspberry and dewberry. It is found in almost every locality where these fruits are grown. While the disease is common, it is not often serious. Rather it is enphytotic, doing but little damage under ordinary conditions. In 1905, however, 20 per cent foliage-losses were reported from Ohio and Florida.

Symptoms.

The appearance of this disease is said to vary to a marked extent on different varieties. On some varieties the spots on the leaves (Fig. 46) are light-brown, while on others they are dark-brown. The lesions are comparatively small, measuring about one-eighth of an inch in diameter. The diseased portion is at first purplish, although a brown color is assumed as the affected tissue dies. In old spots the center is whitish, and the border is brownish or reddish.

Cause.

The causal fungus is *Septoria Rubi*. It belongs to a group of organisms the life-histories of which are fairly well understood.

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But the successive activities of *Septoria Rubi* have not been carefully studied; this is probably due to the fact that the disease which it produces has never been of great economic importance.

The mycelium of the fungus growing in local areas within the leaf-tissues causes the color changes and finally their death. The fungus forms fruiting bodies — pycnidia — just beneath the upper surface of the cuticle. These, as they mature, break through the cuticle and stand exposed over the affected area. Within the pycnidia spores are developed which at maturity, under moist weather conditions, ooze out and are scattered to other leaves. Here they germinate in water and the result is a germtube which in some way gets into the leaf-tissues. A spot is the outcome. Little is known of the winter condition and of the manner in which the first infections in the spring are brought about.

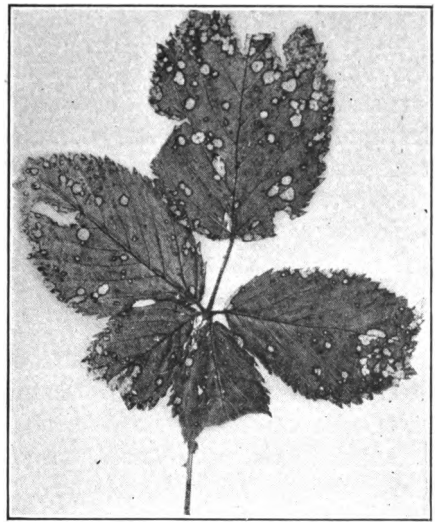

FIG. 46. — Blackberry leaf-spot.

Control.

In cases in which the leaf-spot disease becomes a menace it may be kept in check fairly well by the use of bordeaux mixture, 4–5–50, or lime-sulfur 1–50. The spray should be applied as follows: (1) shortly after the leaf-buds unfold, (2) subsequent applications at intervals of two to three weeks until the fruit is about two-thirds grown.

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As a rule the disease is not of sufficient importance to warrant spraying for its control.

Yellow Late-Rust

Caused by *Kuehneola albida* (Kuehn) P. Magn.

This is a disease peculiar to wild and cultivated blackberries. It is found in Europe and in eastern North America. In the northern United States where it occurs there is no need for alarm on account of its late fall appearance. However, it may assume a more important rôle south of New York State.

Symptoms.

Plants in sandy soil are said to be more affected. Rust pustules are produced on the canes the last of May or in June, or about blossoming time. These are bright, lemon-yellow in color, and measure from one-eighth to one-half an inch in diameter. They are usually found on the stem near the ground. Later the leaves are affected, the disease first appearing on the lower surface of the leaves of fruiting canes, then on those leaves attached to the new canes. The fruit-stalks and flowers are also rusted.

The general appearance of the disease has given rise to the names yellow late-rust, white-rust, cane-rust and late-rust.

Little is known of the seasonal cycle of the causal fungus, *Kuehneola albida*, and of its control.

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Blue-Stem

Caused by *Acrostolagomus caulophagus* Lawrence

Blue-stem is better known on raspberries than on blackberries. Little trouble has been reported from this disease on the latter fruit. It is a Pacific Coast disease at present. (See Raspberry, page 416.)

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CHAPTER V

CHERRY DISEASES

THERE are several diseases of the cherry tree which, if left uncurbed, may prove a serious menace to the growing of this fruit. Most cherry diseases, however, yield rather promptly to control measures — a point very encouraging to cherry-growers. But brown-rot often runs rampant, doing considerable mischief. Other conspicuous and injurious troubles affecting the cherry are leaf-blight, powdery-mildew and black-knot. These along with several other cherry diseases are common to the plum and peach.

BROWN-ROT

Caused by *Sclerotinia cinerea* (Bon.) Schröt.

Cherries are subject to the same brown-rot that affects peaches and plums. The trouble is extremely common on all these fruits, although perhaps the cherry is less seriously damaged than peaches and plums. White sweet cherries are more susceptible than sour and native cherries. The general statement is also made by authorities that the juicy, best-flavored varieties are more commonly affected than others. The Heiderman sand-cherry and Governor Wood both are listed as especially susceptible to brown-rot; on the other hand, the Montmorency is relatively resistant. Susceptibility generally increases as cherries approach maturity.

Brown-rot is the most destructive disease of cherries. It

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may kill the blossoms, thus destroying the set of fruit. It may destroy a whole crop of green, ripe or ripening fruit (Fig. 47) before picking. The trouble is particularly destructive in transit and in the market. Sometimes leaves (Fig. 47) and twigs are blighted. The losses involved are frequently extensive. The disease commonly prevents choice cherries from being placed on distant markets. Losses have been estimated from 10 to 50 per cent. Its wide range over the globe and the numerous fruits which the brown-rot fungus attack are

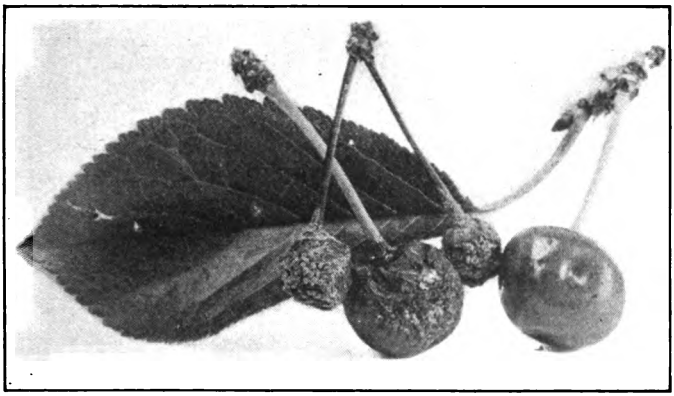

FIG. 47. — Brown-rot on cherry.

factors in the economic importance of the disease. The disease is readily controlled by spraying with lime-sulfur, diluted 1–40; first application, just before the blossoms open to prevent blossom-blight; second application, just after the shucks have fallen, when fruits are about the size of peas; and again as the fruit begins to color. Arsenate of lead, 2 pounds to 50 gallons, should be added to the second application for worms. Finely ground sulfur and dry arsenate of lead 90–10 may be dusted on the trees instead of spraying them. (See more detailed discussion under Peach, page 270.)

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LEAF-BLIGHT

Caused by *Coccomyces hiemalis* Higgins and *C. lutescens* Higgins

It is probable that this disease is of foreign origin, the first report coming from Europe in 1884. Shortly thereafter the trouble became common in the United States. Special attention was given to the disease about 1890, at which time it was fairly well known to American nurserymen. It undoubtedly now prevails in all regions of the globe where the cherry is under cultivation, and is a pest both in the nursery and in the orchard. Sweet cherries, particularly mazzard seedlings, are very susceptible, whereas mahaleb seedlings are usually less so. It may be that the absence of the proper species of the pathogene accounts for the escape of the mahaleb seedlings. Early varieties are often much less affected than later-maturing ones.

The damage caused by the cherry leaf-blight disease is not easily measured. It is believed to be one of the most important diseases affecting cherries in the nursery. Under conditions most favorable to the development of the causal pathogene the most susceptible types are completely defoliated. This results in an early loss of vigor by the young trees. Such trees are not able to mature their wood properly, and thus they withstand the dormant period with more difficulty and they

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enter the next season with decreased vitality. A case is on record where 40,000 young cherry trees were lost on account of leaf-blight alone. The loss in Ohio in 1905 is estimated at $25,000. The preceding year it is estimated to have caused a loss of 8 per cent, in Maryland. One nursery company in

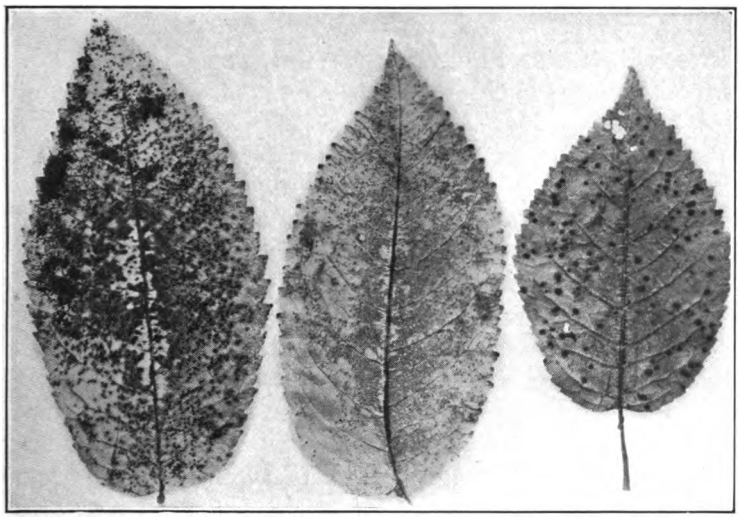

FIG. 48. — Leaf-blight (yellow-leaf, or leaf-spot) on sweet cherry; types of lesions on upper and lower surfaces. Center leaf shows whitish masses (spores) of the pathogene.

Nebraska claims to have lost $40,000 in 1903 on account of this disease. These examples serve to show the possible destruction which may be wrought by leaf-blight.

Symptoms.

The fruit and pedicels are liable to show the disease, but the foliage (Fig. 48) is by far the most common seat of the trouble. Toward the last of May or early in June affected leaves exhibit slightly discolored, dark-blue areas on the upper surface (Fig. 48, left). These are not more than one-eighth of an inch in

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diameter, and they may be scattered over the whole blade or confined to one portion. Within a week or so the affected tissue becomes dark-red or reddish brown in color. Later developments may be one of two types; either the affected portions drop out, leaving circular areas in the leaf, or the whole leaf turns yellow (Fig. 48, center). A single leaf may show both types of symptoms, but with cherries, the yellowing of the leaves is the more common, whence the common name yellow-leaf. An allied trouble affects plums, and while the leaves show both a yellowing and a shot-hole effect, the latter symptom is the more common. During periods of wet weather whitish masses appear on the lower surface of the leaf-lesions (Fig. 48, center). Sometimes these pustules are found in the center of the spot on the upper surface. Any time after the last of June premature defoliation is likely to occur on affected trees.

Affected pedicels show spots one-fourth of an inch or less in length, which extend one-third or more of the way around, often girdling the pedicels. The presence of such lesions causes the fruit to ripen unevenly. Lesions on the fruit are unusual and probably are never important. They manifest themselves in the form of dead, brown spots with whitish fruiting bodies in their centers.

Cause.

The leaf-blight of sweet cherry is caused by *Coccomyces hiemalis*, a fungus with a not unusual life-history. The leaf-blight on mahaleb seedlings is caused by a very similar organism, *Coccomyces lutescens*. These fungi hibernate in the old fallen leaves as sexual structures called apothecia. In the spring ascospores are ejected from the apothecia, and are carried by the wind to the new foliage on which spore-germination occurs. About a week or ten days later signs of leaf-blight are visible to the naked eye. The reader will recall that this has occurred by June 1 at least. The spore on germination sends its tube into the young leaf and mycelium rapidly develops. The

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mycelial threads grow between the leaf-cells, but send haustoria into the cells, thus obtaining food for the invading pathogene. Soon the cells of the affected portion are killed, and they exhibit characteristic color-changes, as already described under Symptoms.

During the summer asexual spores are developed. The mycelium amasses near the surface of the leaf and there results a fruiting body — an acervulus. From the interior of this structure conidia arise in great numbers. They push out on the leaf in large quantities, finally piling up until they are visible to the naked eye as whitish masses. These spores are carried by natural agencies to other leaves, where they germinate, and infection results in a manner similar to that described above. As new growth appears it is exposed to infection throughout the growing-season. When the leaf falls, the fungus is carried with it. Here on the ground, within the leaf-tissues, it passes the winter as apothecia formed from the mycelium in late autumn.

Control.

The elimination of the old leaves and protection of the developing leaves during the growing-season constitute the known measures of control of leaf-blight. Plowing under fallen leaves removes a large portion of the source of the inoculum. This practice, although highly commendable, is not reliable in itself as a means of combating this disease. A few leaves always remain on the surface of the soil and are sufficient to cause trouble.

The healthy foliage may be protected by the application of a sulfur fungicide. For the past few years lime-sulfur solution has been used, but recently it has been shown that sulfur dust is very satisfactory and effective and may soon replace the liquid fungicide. Bordeaux mixture, 5–5–50, is also used. In treating orchard trees the applications should be made as follows: (1) when the fruit is free from the calyx; (2) two weeks later; (3) just after the fruit has been picked; (4) three weeks later,

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if necessary. If lime-sulfur solution is used, the strength advised is 1 to 50, with iron-sulfate one and one-fourth pounds to fifty gallons of the diluted solution. This mixture is used with safety, particularly with the addition of iron-sulfate, which reduces the burning qualities and increases adhesiveness. Sulfur dust, used at the rate of ninety parts finely ground sulfur to ten parts powdered lead arsenate, has been found to be effective.

In the nursery the same fungicides are recommended. The first application should be made when the cherry buds are about eight to twelve inches high. Subsequent applications depend on the weather. As a rule, five to seven applications at intervals of two weeks are sufficient. The earlier applications often can be made advantageously at shorter intervals in order to keep the new growth covered.

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Powdery-Mildew

Caused by *Podosphæra Oxyacanthæ* (Fries) de Bary

It is chiefly the young leaves and the tips of branches of young cherries that suffer from the effects of powdery-mildew. But the disease is commonly observed also on mature trees. Furthermore the mildew pathogene attacks other fruit-trees like the apple, plum, peach, crab-apple, quince and persimmon, as well as certain shrubs, including juneberry, hawthorn, spiræa and huckleberry.

Cherry powdery-mildew was first described in France about 1800, on hawthorn. Subsequently it was discovered in Germany on the cherry. In America the disease is very common and widespread in the eastern and central states, and is reported from the Rocky Mountain states and California. It also occurs in Canada.

The pathogene shows preference for budded sour cherry stock, although sweet varieties and mazzards are known to suffer from its attacks. The mahaleb cherry and the variety Governor Wood, a sweet variety, have exhibited a certain amount of resistance, while the Hoy variety has been noted as free from mildew in cases where other varieties succumb.

Symptoms.

Powdery-mildew is usually found on the young sprouts and at the tips of branches, where it affects both the foliage and the wood (Fig. 49). It may be observed as early as the first of June, but does not develop sufficiently to attract attention until July, and it increases in prominence throughout the late summer and early autumn. Affected parts show small, round, whitish blotches, having a radiating appearance. These blotches spread and coalesce so that a considerable portion of an affected leaf or twig becomes covered by a white felt. These mildewed spots soon exhibit black spherical bodies which are scattered

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over the affected area. The internodes of diseased twigs are shortened and thickened (Fig. 49, right). Both surfaces of the leaves are liable to attack, although as a rule only one side of a single leaf shows mildew at a given time. Diseased leaves are caused to curl inward and upward in a very marked fashion.

Cause.

The pathogene is a fungus known as *Podosphæra Oxyacanthæ*. It thrives best during warm, dry weather; therefore outbreaks

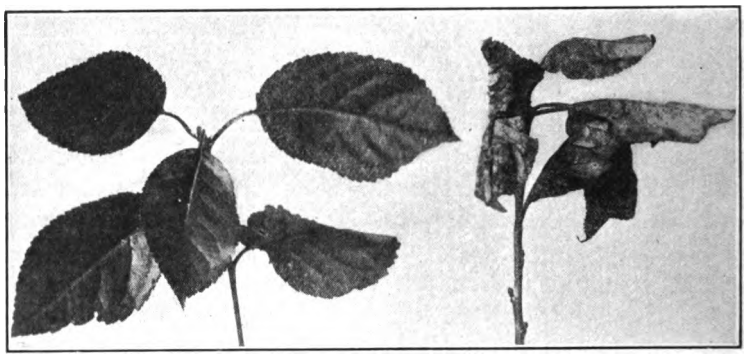

FIG. 49. — Powdery-mildew of cherry; healthy shoot on left, diseased on right.

occur in seasons characterized by such conditions. A drought in the Mississippi Valley in 1887 and 1888 was accompanied by an epiphytotic of cherry-mildew.

The fungus hibernates as mature perithecia. In the spring ascospores which develop within these bodies escape by the disintegration of the perithecia. Those ascospores which fall on susceptible parts germinate in the presence of moisture, the germtube developing into mycelium which grows over the surface of leaves and twigs. This growth becomes profuse and constitutes the mildew so noticeable as a symptom of the disease. None of the mycelial threads enters the tissues of the affected plant, but small suckers, called haustoria, arise as branches

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from the hyphæ and penetrate the outer cells of the leaf or twig. As development of the fungus proceeds, conidiophores grow erect and bear chains of conidia. These spores in mass give the powdery aspect to the lesions. At maturity the conidia are carried by the wind to other leaves and twigs, where new infections are initiated. Later in the summer perithecia begin to develop. These are yellowish at first, later brownish, and finally black. When mature, each perithecium contains a single ascus, which contains eight ascospores.

Control.

The application of some standard fungicide is an efficient remedy for powdery-mildew of the cherry. It is recommended that the orchard be sprayed with bordeaux mixture, or sulfur dust, making the first application as soon as the disease appears. Repeat the application ten days later, if necessary. Sulfur dust may also be used with good success in the orchard. Sulfur is preferable in the nursery. Lime-sulfur 1 to 50 applied as directed for the orchard has proved satisfactory. Add 3 pounds of iron-sulfate to each 50 gallons of the spray mixture; this procedure increases adhesiveness and decreases the caustic qualities of the fungicide. Sulfur dust 90 parts, and powdered lead 10 parts, has proved satisfactory and effective, and may be substituted for lime-sulfur solution. Applications subsequent to the first may number two to four, depending on the severity of the disease. As a rule the schedule for the control of cherry leaf-blight keeps mildew under control (see page 175).

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Black-Knot

Caused by *Plowrightia morbosa* (Schw.) Sacc.

Black-knot is perhaps the most conspicuous disease of cherries. It is common also to plums; in fact, plums suffer more from this disease than do cherries. However, cherries of many varieties are affected with black-knot, including choke-cherry, wild black or rum-cherry, bird or pin-cherry, and morello varieties. Sweet cherries are said to be much less affected than sours. The English Morello, the standard late sour cherry of North America, probably suffers as much as any of the cherries. At times serious injury is inflicted on this and other varieties, particularly in the eastern United States. In North Carolina, for example, cherry-growing was abandoned in 1906 because of black-knot injury. The disease also occurs in the West. (For fuller discussion of symptoms, cause and control, see Plum, page 356.)

Frost-Injury

Caused by the action of low temperatures

Cherries are injured more or less every year by frost in localities away from large bodies of water. The sweet varieties are particularly susceptible to frost-injury, although sour cherries are affected. Gum exudes from the injured bark in early summer, and on cutting into such places large gum-pockets are revealed. (For fuller discussion, see Apple, page 35.)

Die-Back

Caused by *Valsa leucostoma* Fr. var. *cincta* Rolfs

This disease is common on stone- and pome-fruits everywhere. Considerable difficulty has been experienced in some sections

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of Germany because of the severe injury inflicted on cherry trees. In the United States both the wild and cultivated cherries are affected. The reader is referred to Peach for a description of the symptoms, cause and control, page 300.

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BACTERIAL-GUMMOSIS

Caused by *Bacterium Cerasi* (Griffin) = *Pseudomonas Cerasus* Griffin

The phenomenon of gum-flow is common to stone and citrus fruit-trees. It results from stimulation produced by foreign factors of one kind or another. The flow of gum, gumming, or gummosis, is not a disease, although in many cases it is a sign of disease. In other cases it is an indication of injury. The term gummosis, then, is used broadly to designate any disease or injury which is accompanied by a gumming, or a flow of gum. The remarks presented here refer in most cases to a particular bacterial disease of the cherry, plum, peach and apricot which is commonly accompanied by gum-flow. So far as is known at present this disease, called bacterial-gummosis of cherry, occurs only in western Oregon and Washington. Time may show, however, that this bacterial-gummosis prevails in other sections of the United States. It is to be noted in this connection that the flow of gum in the cherry in the states already referred to is frequently caused by factors other than the bacteria under consideration. In the Pacific Northwest the disease is usually called cherry-gummosis, but the common designation bacterial-gummosis is preferable in that it is more specific.

Among the plants affected by this disease the cherry is most

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susceptible. Sour cherries are slightly or not at all attacked. Sweet cherries, on the other hand, are very susceptible to bacterial gummosis. The Bing and Napoleon (Royal Ann, Ox Heart), two very desirable cherries in the Northwest, suffer considerably from bacterial-gummosis. Certain sweet varieties, however, like the Lambert and May Duke, are comparatively resistant.

Symptoms.

This disease is most serious on trees which have been set for three or four years. In many cases they may be killed. On trees more than ten or twelve years old the damage is largely a blighting of the smaller branches, spurs and buds, and it is usually not serious.

Often there is little indication of the disease until whole trees or branches fail to leaf in the spring, or until there is a sudden wilting in the growing-season. Careful examination of cherry trees showing such symptoms will usually reveal a girdling of a limb or trunk. Sometimes there is no gumming, but ordinarily a more or less copious flow of gum accompanies such a condition (Fig. 50). The amount of gum exuded from lesions is no indication of the amount or severity of the disease. On cutting into the bark at affected points the inner tissues will be found to be brownish and the appearance will indicate approaching death. A sour odor may accompany this condition. The lesion produced on the woody parts is a canker. At first the bark is discolored. The discolored area enlarges and the surface of the bark becomes depressed. Then gum exudes from the margin of the canker. Subsequently the extension of the canker ceases and a callus develops about its margin. Enlargement of the cankered area takes place in the winter and early spring, but ceases by the last of March. The callus is formed in the spring and summer. The next winter the callus may be attacked and the lesion extended. In the spring a second callus is formed. This process may be repeated annually

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for several years. Finally an affected branch or trunk may be girdled, as a result of which the foliage becomes yellow, then brown, and finally withers and dies. Such foliage hangs on the tree for some time. Below a girdled trunk, suckers may spring up from the healthy part.

In addition to the cankers just described another phase of bacterial-gummosis is found in a blighting of the spurs and buds. The first indication of this is usually not noticed until

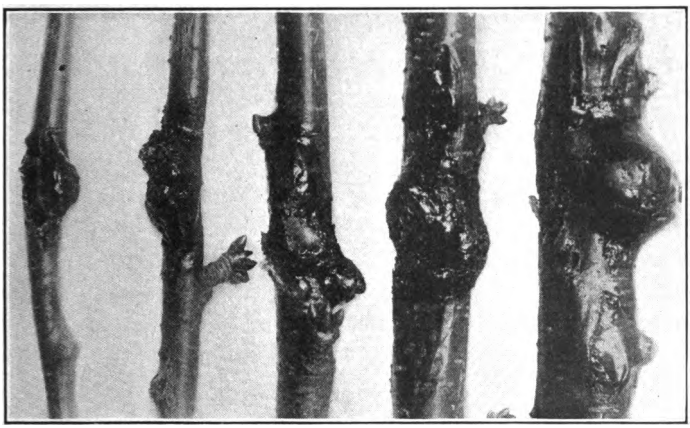

FIG. 50. — Cherry twigs affected with bacterial-gummosis; cankers about the base of blighted spurs. Gum-flow evident.

spring, when some of the buds fail to swell and open when others unfold. A drop of gum often exudes from such buds. Sometimes affected buds do unfold in the spring in an apparently normal fashion, but before the blossoms open, these buds wilt and become dry.

Cause.

The bacterial-gummosis of cherry is caused by *Bacterium Cerasi.* Before proceeding with a discussion of this particular disease, brief reference will be made to the causal nature of gummosis in general. As already indicated, the trouble may be

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due to one or more of several factors. These factors act in a stimulative manner. Among such causal agents may be noted: (1) mechanical and insect injuries; (2) unfavorable soil and climatic conditions; (3) fungi; (4) bacteria.

Mechanical injuries such as those produced by bruising, or by cultivating tools, may induce gum-flow. Pruning wounds often exude gum. Insects, such as borers, produce injuries through which gum commonly exudes.

The relation of soil and climatic conditions to the formation and flow of gum is not well understood. Trees in low places where the soil is excessively wet are often more subject to gummosis than others. On the other hand, trees on well-drained soil may exude gum. Not infrequently frost induces gummosis.

Fungi are common causal agents in gum-flow. Some of the more important ones will be noted: (1) the blight fungus, *Coryneum Beijerinckii;* (2) the die-back pathogene, *Valsa leucostoma;* (3) the black-knot fungus, *Plowrightia morbosa;* (4) the brown-rot fungus, *Sclerotinia cinerea;* (5) the root-rot fungus, *Armillaria mellea.* Doubtless many other fungi induce gumming.

It will be seen that gummosis is associated with a variety of conditions of the plant, but it is often difficult or impossible to attribute a given case of gum-flow to any one cause. Gum is formed internally and may not always exude. It is formed in pockets which are not visible on the surface of the bark. Usually, however, the bark ruptures and the gum flows out.

With reference to bacteria as the cause of gummosis, the development of *Bacterium Cerasi* will now be considered. Many facts in the life-history of the organism are lacking. The bacteria apparently lie dormant in the bark through the late spring, summer, fall and early winter. In the case of limb and trunk cankers the bacteria may or may not become active

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again. In the event of renewed activity in the winter the bacteria spread at the edge of the old cankers, thus enlarging these lesions. The bacteria probably lie dormant also in the buds. The effect of their action is not ordinarily observed until the buds fail to open in the spring or until, after opening, they suddenly die. From the cankers, bacteria may possibly be carried by insects in the summer to new points where infections result. The bacteria attack the outer bark, then the phloem and cambium. These affected elements all turn brown. Brown streaks are found in the bark (between the phloem and outer cortex) extending above and below a canker. Gum pockets are formed under the bark, which splits and allows the gum to ooze forth.

The theory of gum-formation has created no little interest. It is now generally held that gum is formed through the transformation and liquefaction of the walls of certain cells. Such cells are formed abnormally by the cambium as the result of stimulation by parasites or other factors already enumerated. In bacterial-gummosis of the cherry this stimulating factor is *Bacterium Cerasi*. Probably it produces an enzyme which dissolves the walls of the cells, with the result that gum is formed in pockets made in turn by the dissolution of the cell-walls in a local area. In order that gum may be produced, an abundance of water seems necessary. It also seems essential that the tree be in a growing condition for gum-production. It may then be understood why young trees exude gum more often than do old trees.

Control.

Recommendations for the control of the bacterial-gummosis of the cherry are made in the Northwest along three lines: (1) the removal of cankers; (2) the protection of susceptible trunks and limbs with coarse cloth or burlap; (3) the use of resistant seedlings and the growing of resistant varieties.

Cankers on old trees are rare, so that surgical methods apply

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to young trees only. The removal of diseased and dead bark has several advantages. Such operations should prevent the further spread of the bacteria in a given canker. The elimination of dead bark from cankers in which the bacteria have ceased activity will permit more rapid healing of the wound. The removal of diseased bark also means the removal of a source of the trouble. The trees should be inspected late in the winter and early in the spring for new infections. All discolored bark should be removed as advised for fire-blight cankers (page 23). All wounds should be disinfected with corrosive sublimate 1–1000, and a wound-dressing should be applied. These measures should give effective results.

It is suggested that trunks and limbs be wrapped with burlap or coarse cloth until the young tree passes the danger-point. This method is as yet in the experimental stage and should be used with this fact in mind.

The use of mazzard seedlings on which desirable varieties may be grafted is strongly advised. These stocks show striking resistance to gummosis. The buds should be set in the limbs and not in the trunks; this prevents the spread of the bacteria from one limb to another. The grower is cautioned concerning mazzard seedlings: various sweet cherry seedlings are probably sold under the name of mazzard. Some growers raise their own seedlings. The feasibility of this practice must be determined by the grower. (See also general discussion of Gummosis under Peach, page 303.)

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Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

The crown-gall disease is so cosmopolitan in its host-range that the cherry could not be expected to escape in all cases. It is the younger trees that suffer, particularly if the knots, or galls, surround the crowns. In the North, at least, it has been observed that little injury is done to mature trees. Sometimes trees planted with galls may outgrow the disease in a year or so. It is not safe, however, to assume that this will always occur; affected trees should not be used in plantings. (See fuller discussion under Apple, page 108.)

Armillaria Root-Rot

Caused by *Armillaria mellea* (Fries) Quel.

Among the stone fruit-trees affected by this root-disease the cherry stands out as one of the most resistant. In the Pacific Northwest, however, a few cases of root-rot caused by *Armillaria mellea* have come to the attention of growers and plant pathologists. Special discussion is not necessary here; see Apple, page 96.

Leaf-Rust

Caused by *Puccinia Pruni-spinosæ* Pers.

On the leaves of many wild and cultivated cherries there is frequently a rust-disease. It is said to occur more commonly in the eastern United States. The same disease is found on the peach and on wild and domesticated forms of the plum. It is variously known as plum leaf-rust, prune-rust, peach-rust and leaf-rust of cherry. (See fuller account under Plum, page 377.)

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Witches'-Broom

Caused by *Exoascus Cerasi* (Fckl.) Sadeb.

Witches'-broom is a peculiar type of gall in which there is an over-production of whole organs, resulting in a broom, or nest-like habit of growth. This name is in general use in North America. In England, the disease is called thunder-bushes, bull-boughs, bull-wood and bull-branch. The Germans refer to it under the name Hexenbesen, while the French call it Balai du Sorcière.

The disease is very common and destructive in Europe, where both sweet and sour cherries are affected. Although the English sweet cherry (*Prunus avium*) is commonly cultivated in eastern United States, the disease was not recorded until 1886, when it was reported from Germantown, Pennsylvania. About ten years later it was found in Long Island at scattering points. The witches'-broom disease of cherry is not a disease well-known to American cherry-growers. It occurs sparingly in New York, but is said to be common in Oregon. It has also been observed in a few other states, including Washington, Minnesota, Pennsylvania and New Jersey. The disease is of most interest perhaps because of the fact that the pathogene spreads so slowly, and on account of the peculiar effect on the cherry. Its history thus far in this country does not indicate that the disease should ever be feared by cherry-growers. Records show that the disease may affect in this country the following cherries: English sweet cherry (*Prunus avium*), sour cherry (*Prunus Cerasus*), wild black cherry (*Prunus serotina*), wild red cherry (*Prunus pennsylvanica*), choke-cherry (*Prunus virginiana*). Several varieties and species of plums are also affected.

Symptoms.

The twigs and leaves are susceptible to this disease. On the former it produces a very striking deformity, although not a

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killing, of the affected parts (Fig. 51). Infected branches become more numerous and are more or less elongated (Fig. 51). Authorities disagree on the matter of whether affected twigs are thickened. In some cases the twigs become so numerous that they are not able to bear their own weight, and as a result the abnormal portion of the tree droops. In such cases the tips of the branches usually turn upward (Fig. 51). The over-production of branches at a local region results in a broom-like growth. Some of the brooms are large enough and so conspicuous that they are very noticeable from a distance. This is especially true when the tree is bare of leaves. These broom-like growths are also conspicuous at blossoming-time; for they bear few or no flowers, hence the affected portion stands out in bold contrast to the remaining blossoming-branches. Leaves also come out on brooms before they do on healthy branches.

FIG. 51. — Witches'-broom on cherry.

Affected leaves take on a crinkled shape and a reddish discoloration. The disease on leaves is usually referred to as leaf-curl, and the abnormalities exhibited are very much like those of the peach leaf-curl (see page 277). Affected leaves fall prematurely and later new foliage appears. On their lower surfaces a whitish coat may be found prior to defoliation.

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Cause.

The causal fungus, *Exoascus Cerasi*, is very closely related to the fungus causing peach leaf-curl and to that of plum-pockets (see pages 277 and 373, respectively). Its full life-history is not known. The mycelium invades the twigs, living in them from year to year. Both the bark and wood are attacked. The pith-cells, medullary-ray cells and outer cells of the bark (hypodermis) are greatly increased in number as a result of stimulation set up by the parasite. The sclerenchyma fibers, on the other hand, are fewer or lacking. The stimulating effect of the fungus brings about a condition whereby, instead of flower-buds being produced as in ordinary cases, abnormal twigs are developed, giving the broomy aspect already described. The mycelium invades the leaves, causing them to curl and fall. Finally, before defoliation, the fungus forms a fruiting layer of asci with ascospores on the lower surface of the leaves. These bodies give to this surface the whitish appearance already mentioned.

Control.

Fortunately the disease is not usually of sufficient importance to require urgent attention. Little experimental work has been done on which to base recommendations for American conditions. Since the brooms bear neither flowers nor fruit and are a source of trouble, they should therefore be removed. The cut should be made several inches below the lowest point in the diseased portion. It is said that this method readily controls the disease.

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Fire-Blight

Caused by *Bacillus amylovorus* (Burr.) Trev.

It has been recently shown that fire-blight, so common on pear, apple and quince, also affects the cherry in the Pacific Northwest. As yet, however, it is not very prevalent on this fruit and the damage thus far is comparatively slight.

The disease appears at first as small pits in the surface of the fruit. The lesions are few for a time, but gradually their number increases until they occur over practically the whole surface. As a result the cherry becomes shrunken and wilted. Amber-colored, sticky drops of bacterial ooze, similar to those appearing on blighted pear fruits, are sometimes observed on affected cherries. (Detailed discussion of Fire-Blight under Pear, page 323.)

Reference

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Scab

Caused by *Cladosporium carpophilum* Thüm.

The scab disease of cherries is most prevalent on the peach, and is found only occasionally on the cherry. The extent of the damage done is believed to be inconsiderable. Olivaceous to brownish spots appear on the fruits shortly before the ripening period. Records of it come from Iowa and Minnesota. More detailed discussion of the disease caused by this fungus is given under Peach, page 294.

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CHAPTER VI

CRANBERRY DISEASES

THE centers for the production of cranberries in the United States are New Jersey, Massachusetts and Wisconsin. So far as information is available, it appears that diseases of the cranberry are coincident with cranberry-culture. The bog is necessarily moist, which condition is highly favorable to the growth of parasitic fungi. The more important diseases are scald, gall and hypertrophy.

SCALD

Caused by *Guignardia Vaccinii* Shear

The late varieties of cranberries suffer from a disease known as rot or scald. The term scald has been in general use for a long time, and it owes its origin to a belief of the growers that the softening of diseased fruit was actually a scalding caused by heat from the sun when the berries were wet. The name scald as used by many growers embraces at least three diseases of the fruit: scald, rot and anthracnose. The discussion here concerns only the true scald.

Growers have known this troublesome disease for several years. It was the subject of discussions at the early meetings of the New Jersey Cranberry Growers' Association which was organized in 1869 (now known as the American Cranberry Growers' Association). Scald is more prevalent in the East and Southeast than in the Middle West, although it is well

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known to Wisconsin cranberry-growers. It also occurs in Canada and in Europe.

The amount of damage done by this disease varies considerably with the season. In some years seventy-five per cent of the crop is destroyed. In New Jersey the annual loss to cranberry-growers on account of scald is estimated at about one-third of the crop.

Symptoms.

The berries, flowers, leaves, stems and roots are affected. The first evidence of scald appears as a minute, light-colored, watery area on the surface of a berry. This enlarges until the whole berry is involved, softens, and turns brown (Fig. 52). It is thought that in the East affected berries do not turn brown, and that this character is influenced by the soil and climatic conditions. Sometimes several spots develop on a single berry. Finally, either the whole affected fruit assumes a scalded or cooked appearance; or, in case only a portion decays, the berry shows a concave surface on the affected side. The interior of scalded berries is soft and watery. It is sometimes difficult to say whether a berry is affected with scald, for although it appears healthy it may still be diseased. Berries when affected before they are one-half grown usually hang shriveled, and covered with black dots — the fruiting bodies of the causal fungus. Dark concentric rings often show; however, this is not a peculiarity of scald, for it does not occur constantly, and furthermore

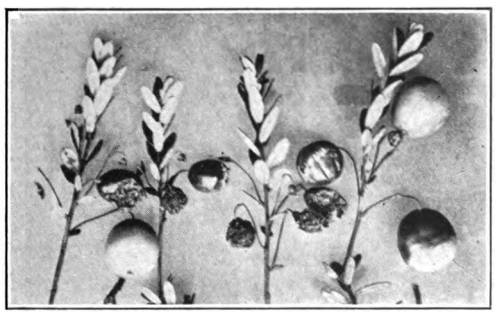

FIG. 52. — Cranberry-scald.

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it is an accompanying characteristic of cranberry-rot (see page 201).

When the flowers are affected, they suddenly shrivel and die. This effect is commonly known as blast.

Leaves are not usually affected, but do not always escape. Brown spots, irregular in outline, are produced at times. Within such areas the black fruiting bodies of the pathogene may be found. Ultimately the leaves turn yellow and fall.

Cause.

Cranberry-scald is caused by the fungus *Guignardia Vaccinii*. It was formerly held that it is caused by too much acid in the soil. It was also believed that excessive heat and drought are contributing factors which induced fermentation in the fruit. A condition similar to scald is sometimes induced when berries are flooded and kept covered with water for a half day or more during hot weather.

The causal fungus, *Guignardia Vaccinii*, is found generally in the cranberry sections of the country. It was once thought that it lived perennially in the stems, and that it entered the fruit therefrom. But now this is regarded as a false idea. The fungus winters in the old fallen leaves. In the spring pycnospores initiate the first infections. The time and manner of this process is unknown. But it seems very likely that it occurs very early in the growing-season, soon after the water is removed from the bog, and while the berries and leaves are quite young. Assuming that pycnospores from old leaves come to lie on the susceptible parts, then, under conditions favorable to the process of germination, a germtube is developed from each spore. The organ inoculated is penetrated and a mycelium is developed. It appears that after the fungus has entered its host it may remain inactive for some time, during which period there is no external evidence of the disease or the fungus. Thus affected berries may pass unnoticed as healthy fruit. The conditions affecting the length of this

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period of inactivity are not certainly known. With the resumption of growth the tissue in the region of the mycelium is killed, and the lesions described above are produced. The fungus fruiting bodies, pycnidia, break through the epidermis, and expose their tips. Each pycnidium contains, at maturity, many pycnospores which coil out in a gelatinous tendril. A second type of fruiting body, perithecia, is sometimes developed, but these occur less frequently, and are not regarded as important in the dissemination of the fungus.

The conditions favoring the fungus are not unusual. Warm, wet weather furnishes the best conditions for maximum development. If berries are kept at a high temperature after picking, the disease is greatly increased.

Control.

It has been shown that bordeaux mixture, 5–5–50, when thoroughly applied, gives satisfactory results. Resin-fish-oil soap at the rate of four pounds to fifty gallons is added as a sticker. Five applications are recommended. In general, not more than fifteen days should elapse between any two applications. The first should be made early in June; the second later in June when the blossoms are ready to open; third, as soon as the plants have passed the height of their blooming period; subsequent applications at fifteen-day intervals.

The evidence at hand indicates strongly that regulation of the water-supply is a very important factor in the control of all cranberry diseases. The amount needed varies with the type of soil and the contour of the land. The supply should be controllable. It should be constant; fluctuations should be avoided during the growing-season. Keeping the plants continuously moist, but not wet, is recommended.

Bog sanitation should be practiced. Dead leaves and vines should be destroyed. This must be done before the spores are discharged, to be effective — at least within a week after the water has been withdrawn from the bogs in the spring.

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It is advised that plants which seem less subject to the disease be selected for propagation.

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Gall

Caused by *Synchytrium Vaccinii* Thomas

Cranberries and closely allied plants are affected by the gall disease. Among these plants may be noted: azalea, sheep-laurel, calfkill, leather-leaf, huckleberry, winter-green and sweet pepper bush. These are particularly affected when growing along the edge of an infested bog.

The disease was discovered about 1886 in New Jersey. For some time it was thought that the trouble was confined to a single meadow in that state, but now it is known to occur as far north as Newfoundland. Badly infested meadows are rendered practically worthless. In 1889 the American Cran-

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berry Growers' Association at its meeting passed resolutions out of which came a state law for the prevention of the spread of fungous diseases of plants. This law included cranberry gall. Since that time the disease seems to have been less destructive, although it is doubtful whether this fact has any connection with the legislation just mentioned.

Symptoms.

The first signs of gall appear about May first, and by July first the disease is well advanced. As the name indicates, galls are produced. They vary in shape, depending on the part affected, although in general they are cup-shaped. They measure from one-twenty-fifth to one-thirty-sixth of an inch in thickness and length. The galls are found on young stems and leaves and even on the flowers and fruit. Their color is reddish, so that the disease has been called red-rust. A badly infected plant presents in its entirety an unusually red color.

Cause.

The gall disease is caused by a fungus, *Synchytrium Vaccinii.* The pathogene is of a low order and possesses no mycelium. Within each gall globose bodies, sporangia, are produced. And within each sporangium a mass of swarm-spores develop in the spring. The sporangia rupture and the spores are liberated. The spores are motile and move about in water very easily; in fact, they are dependent on abundant moisture for their dissemination. Possibly the fungus is carried from one place to another through the air on the feet of birds, or by the wind. It should be borne in mind that while the cranberry is an evergreen, the other hosts, like azalea and huckleberry, are deciduous, dropping their foliage which might drift for long distances over the snow crusts in the winter. April floods spread the fungus rapidly. Should it enter a bog at the head of a stream, the fungus would quickly spread to all plants growing along that stream below the point of entrance.

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Control.

It is advised that bogs be burned over in the early winter. In such an operation the other hosts already listed should be taken into account. It has been suggested that withholding the water and keeping the bogs dry in the winter and spring might keep the fungus in check.

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HYPERTROPHY

Caused by *Exobasidium Oxycocci* Rostrup

Hypertrophy, or false-blossoms, is erratic in its behavior. Some bogs are free from it, while in others every plant may be diseased. The variety Matthews seems especially susceptible.

The disease is rather limited in its range, being known only in Massachusetts. In 1906 it was unusually severe in that state, destroying a large part of the crop in several bogs. Not only the production of fruit is prevented, but the vitality of the plant is exhausted.

Symptoms.

The fruit-bearing and vegetative shoots, runners, are affected. The disease appears usually in the middle of May or soon after the water is removed from the flooded bogs in the spring. The axillary leaf-buds, which ordinarily remain dor-

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mant, are affected, in response to the attacks of the pathogene. They produce short shoots with leaves abnormally swollen and distorted. These leaves are pink or light rose-colored. The affected leaves stand close together and bear a slight resemblance to a flower, whence the name false-blossoms. This name should be avoided, for there is another disease which passes under this designation. In the genuine false-blossom the floral parts are affected; instead of the typical drooping of the flowers, the flower-buds stand erect, remain greenish, and do not set. This is accompanied by a profuse branching of the vines. Until recently the real false-blossom was known as "Wisconsin false-blossom," but in 1914 it was found in Massachusetts.

Most plants are affected before the blossoms have developed, and thus the production of fruit is prevented. Later in the stage of development the fruiting portions of the causal fungus give to the surface of the distorted parts a fine, grayish, powdered appearance.

Cause.

The symptoms just described are changes brought about by the fungus *Exobasidium Oxycocci*. Its mycelium grows within the leaf and stem-tissues, stimulating them to overgrowth. Fruiting stalks, known as basidia, emerge and stand erect on the surface of the affected portion. These are found from May to September, but are most abundant in the earlier part of the growing season. On each basidium four spores are borne. These bring about infections. Further points regarding the life-cycle of the fungus are not well known.

Control.

Since the activities and whereabouts of the pathogene are not clearly understood, it is almost impossible to suggest dependable control measures. No definite remedy is on record.

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Frost-Injury

Caused by the action of low temperatures

Frost is one of the greatest enemies of the cranberry-grower. It is rather erratic in its occurrence, and very difficult for the average grower to predict; hence the damage is done before any precautions are taken.

Danger from frost comes throughout the growing-season. In June, 1903, the yield in Wisconsin was reduced twenty-five per cent, and in early August, sixty per cent.

It is ordinarily supposed that frost-injury comes about by a settling of cold air from the surrounding higher land on the bogs. This, however, is regarded as of little importance. Frost formation is largely dependent on the temperature of the soil, and on heat-radiation from it as influenced by the conditions of the atmosphere. The influence of drainage, sand, the depth and thickness of the vegetation, especially of moss, seem to be controlling factors in the formation of frost, and its effect on cranberries. It has been noticed that even where the general minimum over a bog was 25° Fahr., if the same were well-drained and sanded, it remained unaffected by frost; while in adjacent plots which were in a moist condition and not sanded, frost destroyed every plant.

It is advised that plants be sprayed with water every two minutes during a frosty night. Or possibly the spraying may be done just about sunrise, thus drawing the frost out of them gradually before the heat of the sun strikes them.

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Rot

Caused by *Acanthorhynchus Vaccinii* Shear

This disease was formerly confused with scald. As stated elsewhere, scald has been used broadly to indicate what is now known to be several distinct diseases. Rot was then included under the term scald.

Next to scald, cranberry-rot is the most important fungous disease of this fruit. It is found commonly in New Jersey, and is reported from West Virginia, Massachusetts, Wisconsin and Nova Scotia.

Symptoms.

The basis for the confusion of rot and scald is their close resemblance. The two diseases do look very much alike, both externally and internally. The first external sign of the cranberry rot disease is that of a small, light-colored soft spot on the berry. The whole berry is finally destroyed. Dark, concentric rings, as described for scald, sometimes appear as a character of the lesion.

Cause.

The causal pathogene is the fungus *Acanthorhynchus Vaccinii.* It hibernates in the fallen leaves and fruits. In the spring ascospores are forcibly discharged from asci in the perithecia. The spores are gelatinous and thus adhere to any object with which they come in contact. It is not known just when this discharge occurs, but probably soon after the water is removed from the bog in the spring.

The ascospores germinate in damp air or in water, producing a short germtube, which is terminated by a peculiar, dark-colored, disc-like body, an appressorium, with a lobed margin. The small projections at the margin firmly attach the appressorium to the leaf by dissolving small cavities in the epidermis. From the center of the appressorium arises another germtube, which enters the leaf usually through a stoma. This whole

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process probably takes place in a few hours. The mode of entrance into fruits has not been determined.

Control.

The treatment as outlined for scald gives satisfactory control for the rot disease (page 195).

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Anthracnose

Caused by *Glomerella cingulata* (Stoneman) Sp. and von S. var. *Vaccinii* Shear

Although cranberry-anthracnose was not reported prior to 1907, it has a wide geographical range through the eastern United States. It is a much less injurious disease than either scald or rot (see pages 192 and 201, respectively).

It is not easy to diagnose a case of the anthracnose disease by gross examination of affected berries. They are not uniform in appearance. Berries affected with anthracnose may be very soft or not. The diseased flesh is usually light-colored. Sometimes flesh-colored, sticky masses of spores appear on the lesion; however, this is not a constant character.

Little of importance is known regarding the life-history of the causal fungus.

The disease is often associated with scald and rot and may be controlled by the use of bordeaux mixture as employed for those diseases.

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CHAPTER VII

CURRANT DISEASES

THE currant is affected by but few diseases. Among the more common ones are: Mycosphærella leaf-spot, anthracnose and cane-blight. For the past few years currants, especially black kinds, and gooseberries as well, have attracted the special attention of the plant pathologist on account of the European rust disease. This fungous trouble, while of no direct importance on the currant, is very destructive to the white pine; on the latter host it is known as the white pine blister-rust. To currant-growers particular attention is called to the account of European rust, page 212.

MYCOSPHÆRELLA LEAF-SPOT

Caused by *Mycosphærella Grossulariæ* (FR.) Lind.

Both currants and gooseberries are commonly affected by this leaf-spot. In certain regions, for example New York, the black varieties of currants, such as the Naples, are said to be more resistant than the red and white currants. On the latter kinds the foliage is more liable to drop when affected by this trouble.

The disease was first given special attention in Massachusetts in 1886; five years later it was an object of study in Iowa. It was rather common in New York in 1899 and 1900, but the damage wrought was not serious. The disease ordinarily occurs wherever the currant and gooseberry are grown. In some

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seasons nearly complete defoliation occurs. This loss of foliage interferes with the maturing of both the wood and buds, and as a result the succeeding crop is injured. Perhaps the cumulative effects constitute the worst feature.

Symptoms.

During the month of June, lesions appear as small brown spots (Fig. 53) on the upper and lower surfaces of the leaves. The spots may number few or several to a leaf. Each diseased area enlarges until it has reached a diameter of about one-eighth of an inch, and the center becomes pale with small black fruiting bodies of the pathogene, while the border remains brown (Fig. 53). Older lesions are frequently confluent. From the last of June to August the affected leaves turn yellow and fall prematurely. This happens on red and white currants particularly. The leaf-spot lesions are distinguished from those of the anthracnose by their larger size, sharp outline, and pale, dead center.

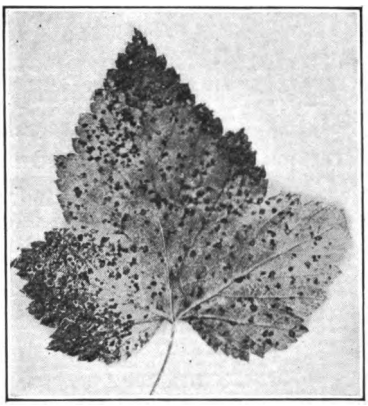

FIG. 53. — Mycosphærella leaf-spot on currant.

Cause.

The causal organism, *Mycosphærella Grossulariæ*, is a fungus which has been known since 1842. It was first described from Europe. When the affected leaves fall to the ground, the fungus is carried with them. It begins to develop perithecia, but these do not reach maturity before winter comes. With the advent of spring these bodies complete their growth. Within each many ascospores are formed. These are discharged with force into the air, are caught by the air currents, and are carried to

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the young leaves. The ascospores germinate and their germ-tubes enter the leaves, wherein mycelium is developed. In a short time the leaf-tissue is killed in local areas and the result is a spot. Finally asexual, summer, fruiting bodies are formed. These are black, spherical structures, called pycnidia, occupying the central portion of the lesion. At maturity each pycnidium is filled with long, curved pycnospores. These ooze out and are scattered promiscuously. Some perchance fall on currant or gooseberry leaves; they germinate and cause spots. Some time during the summer (June to August) the affected leaves fall, and preparations for the winter are again made by the fungus. Damp cloudy weather favors the fullest development of *Mycosphærella Grossulariæ*.

Control.

The spraying schedule for currant-anthracnose is reliable for the control of this leaf-spot. Five to seven applications of lime-sulfur used at the rate of one gallon of lime-sulfur solution (commercial concentrate) to fifty gallons of water are effective. Finely ground sulfur (dust), ninety parts, and powdered lead arsenate, ten parts, has been shown to be satisfactory. The spray or dust should be applied: (1) when the first leaf-clusters appear; (2) subsequently every two weeks until the first of August. (See Control of Currant Anthracnose, page 208.)

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ANTHRACNOSE

Caused by *Pseudopeziza Ribis* Klebahn

This is a disease which appears to some extent every year, and only occasionally does it become epiphytotic. In New York it was serious in 1889 and 1901, and is one of the most common fungous diseases of the currant in Oregon. The trouble is known throughout the United States and Europe. It is also reported from Asia and Australia. Its origin is unknown, but the first mention of it in America comes from Connecticut, having been found there on black currants in 1873. Subsequently it was discovered in the Adirondack Mountains on the fetid currant, in Iowa on the red currant, and is now known to affect several other kinds of currants — both red and white. Gooseberries are also subject to the disease.

The injury from anthracnose, also known as leaf-spot and leaf-blight, comes from its effect upon the leaves and fruit. Plants are known to have been completely defoliated by July 10, and a loss of one-half to two-thirds of the crop of fruit is on record. This early defoliation interferes with the proper ripening of the wood and the formation of fruit-buds for the next year. The disease also affects unfavorably the quality of currants for wine. Old plantations are said to suffer more than young ones, yet the disease is of considerable importance in the nursery. In the nursery older bushes usually suffer more than the first-year cuttings, due perhaps to the fact that the young cuttings are planted on ground which is not ordinarily used for currants and gooseberries. Cuttings in close proximity, however, to the older diseased bushes are very likely to become

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affected. Although both red and white currants are susceptible to anthracnose, some differences in resistance are noticeable. It has been observed that the Albert variety is resistant and the Fay and Victoria, growing in close proximity to the Albert, may be seriously affected. Again, the White Grape and Wilder are susceptible, while the Moore Ruby and Perfection varieties are resistant.

Symptoms.

The disease is primarily a leaf-trouble (Fig. 54), although it also shows on the petioles, young canes, fruit-stalks and fruits.

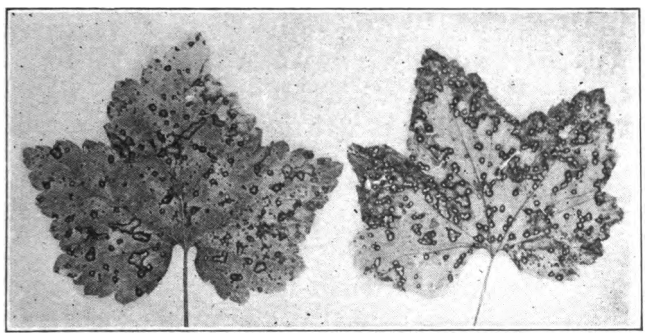

FIG. 54. — Currant-anthracnose lesions on leaves.

The attack is made first on the older leaves, and as a result they become thickly dotted with small dark brown, circular spots, chiefly on the upper surface (Fig. 54). When the trouble assumes large proportions, the leaves turn yellow and fall prematurely. On the berries the spots are small, resembling fly-specks. On the fruit-stalks, the lesions are larger and may be one-fourth to one-half an inch in length and may extend halfway around the stem. On the leaf-petioles, conspicuous, black, slightly sunken spots are formed. The anthracnose is sometimes confused with leaf-spot (see page 203), but can readily be distinguished from it by the smaller size of the spots.

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Cause of anthracnose.

This is a fungous disease, the cause of which is *Pseudopeziza Ribis*. The fungus is carried through the winter by either the conidial or the sexual stages and possibly as mycelium in the canes. The infected leaves fall to the ground in the autumn, and the fungus then penetrates all the tissues. Later it develops an apothecium. In the spring ascopores ripen and are carried to the new leaves, probably by the wind, although no observations have been made on this point. The time of inoculation is unknown, but severely infected plants have been seen on June 8. It is known that about two weeks are required for germination, penetration and the production of visible signs of the disease, from which it may be reasoned that inoculation occurs about the middle of May. The germtube within the leaf, or other susceptible part of the plant, develops mycelium which grows in localized areas and kills the tissues, resulting in the formation of the lesions already described. The mycelium finally forms a mat near the center of the spot where an acervulus develops. At maturity this structure ruptures the epidermis, liberating the conidia in gelatinous masses. These spores are disseminated by rain and insects, but, on account of their gelatinous character, probably are never carried by wind. The conidia continue to propagate the pathogene throughout the growing-season, thus accounting for the extent of the disease in a local area. Conidia produced in late summer are capable of living through the winter. Although, as previously stated, mycelium in the canes may possibly tide the fungus over winter, yet there is considerable evidence to the contrary.

Control.

It has been conclusively shown that bordeaux mixture 5–5–50, or lime-sulfur solution 1–40 or 1–50, when applied at the right time, is effective in the control of anthracnose. It has recently been found that dusting with the sulfur-lead mixture (90 parts finely ground sulfur to 10 parts powdered lead ar-

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senate) is equally effective. It is essential to make the first application of the fungicide before the discharge of any ascospores; as previously noted, this occurs during the middle of May. It is further essential to keep the foliage protected throughout the summer. Make the first application when the leaves are unfolding; apply the fungicide at intervals of ten to twenty days until five or six sprayings have been made. The interval may be lengthened and the number of applications reduced in dry weather. In a wet season more frequent applications are necessary.

Since the fungus hibernates in the fallen leaves, it seems logical that these should be destroyed by plowing or some other means. However, there would in all cases be enough leaves to act as an abundant source of trouble.

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Cane-Blight

Caused by *Botryosphæria Ribis* Grossenbacher and Duggar

Currant cane-blight or necrosis, also known as wilt and blight, affects chiefly the cultivated varieties of the red currant

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(*Ribes vulgare*). It is also found on the cultivated varieties of the black currant (*Ribes nigrum*), and the European gooseberry (*Ribes Grossularia*). The Wilder currant is said to be very resistant to cane-blight.

The disease has been more or less under the careful observation of American pathologists during the past quarter of a century. In 1899 it was studied in New York State under the name of cane-blight. More recently (1911) it has again been given detailed attention in New York. It occurs in Delaware and probably elsewhere, although its confusion with another trouble has made the determination of its range difficult. The history of cane-blight shows that it may become epiphytotic in regions where it is established.

Symptoms.

The conspicuous symptoms of cane-blight consist of a sudden wilting and dying of parts or of whole bushes here and there throughout a plantation. This occurs during the summer, at any time while the plants are in leaf. The leaves wilt, turn brown, and die on certain canes or portions of canes. Finally the leaves fall. The affected wood of a cane is killed at a point some distance below the wilted foliage.

Cause.

Currant cane-blight is due to the work of a fungus, *Botryosphæria Ribis*. It attacks the canes, and the symptoms just described result. It was formerly thought that the disease was due to *Nectria cinnabarina*. The fungus *B. Ribis* passes the winter in the young blighted or dead shoots, or in small cankers. With the advent of the growing-season the pathogene invades and blights the parts below. The spores of the fungus, which develop on the affected parts, may be carried to other plants by the wind or the American currant-borer. It is suggested that the beetle's habit of oviposition may have some relation to the dissemination of the fungus. The currant is most easily infected, that is, most susceptible, during its period

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of transition from active elongation-growth to that of the maturation-stages. Within a week or two after the spores of the fungus are deposited upon the currant, signs of disease appear. The bark, wood and pith are invaded by the mycelium. Sometimes the leaves also are attacked. The mycelium may be observed in the pith as a fine, whitish, webby growth.

Shortly after early infection occurs, the fungus forms a simple spore-stage on the withering tips. This is known to pathologists as the Macrophoma-form. Following the development of this stage, from midsummer to autumn, a stromatic form appears; this is called the Dothiorella-stage. This form shows as small, globose, sclerotic bodies on the shoots which have been killed in the earlier part of the season. Toward the end of the summer the stromata of the second or Dothiorella-type, instead of forming pycnidia, develop perithecia. In this condition the fungus passes the winter. The following May and June pycnospores are formed. They are discharged the latter part of June or in early July. At the same time ascospores develop within the perithecia already mentioned. Thus pycnidia and perithecia may be found on old dead canes together.

Control.

At the present time there is no method of treatment for currant cane-blight which can be confidently recommended. It has been shown that summer pruning, that is, the systematic removal of all diseased canes at frequent intervals during the spring and summer, is of no value so far as the control of cane-blight is concerned. Winter pruning is also inefficient, owing to the difficulty of recognizing affected canes while the plants are devoid of foliage. The careless piling of pruned canes along fences should be avoided. Such brush should be burned before the end of May, in order to destroy the fungus.

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EUROPEAN RUST

Caused by *Cronartium Ribicola* Fisch. von Waldh.

This disease, known also as currant felt-rust and white pine blister-rust, is caused by a fungus which was introduced into the United States from Europe a few years ago. The reforestation movement had created a market for young white pine stock, and since American nurseries have not been able to supply this demand these plants have been imported from Europe. Whatever else may be said of the result of this wholesale importation of foreign white pines, it is to be regretted that the blister-rust pathogene has thus been brought into the United States.

So far as the currant is concerned the disease is of little economic importance, since it does not destroy whole bushes. Gooseberries are also affected, but the condition is similar on that host. The chief importance on these plants (Ribes) lies in the fact that the pathogene lives on them, from which it spreads to the five-needle pines. Here young trees are killed in the nurseries and in plantations; older white pines are also severely affected, sometimes being killed by the disease.

The disease is known in practically all Europe, except in

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the Balkan and Hispanic peninsulas. It is especially prevalent in Germany, Denmark, Belgium and Holland. Just when the parasite was introduced into the United States is not definitely known. It was found in Kansas in 1892, but at that time it was not recognized as the pathogene here under consideration. How it reached Kansas is not known. It became known in 1909 that several millions of young white pines had been previously imported from Germany, with which the fungus unquestionably was introduced. Later shipments brought it from France. Although Americans were warned of the appalling injury by the fungus to white pines in Europe, yet it became established here without attracting government consideration. In 1912 the federal government enacted The Plant Quarantine Act, which provides for the regulation and quarantine of diseased nursery-stock; this, however, came too late, as the pathogene had by that time spread to several states in the Union. The disease has been found in Indiana, Ohio, New York, Pennsylvania, New Jersey, Connecticut, Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Wisconsin and Minnesota. It is also found in Ontario, Canada. It is now widespread on wild and cultivated Ribes in Connecticut, Rhode Island, Massachusetts, New Hampshire and Maine. In time the disease will doubtless be found in all parts of temperate North America, although the federal and state governments are cooperating in an attempt to prevent such a calamity.

Symptoms.

On currants (Figs. 55 and 56) and gooseberries the disease is found from June first to leaf-fall. Rust pustules one-sixteenth to three-eighths of an inch across are formed on the lower sides of the leaves. These are scattered or dense, depending on the intensity of the infection (Figs. 55 and 56). If severely affected, the leaves die and fall prematurely. The rusty mass on the leaves has given rise to the names currant-rust, felt-rust and currant felt-rust.

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The disease may first be observed on the white pine (Fig. 57) in the spring from April to early June. The stem or branches are often girdled and the portion above dies. Most young trees die in a relatively short time; others live for some time, but even old stems finally succumb, the tree eventually breaking at the lesion. The disease in its early stages shows peculiar

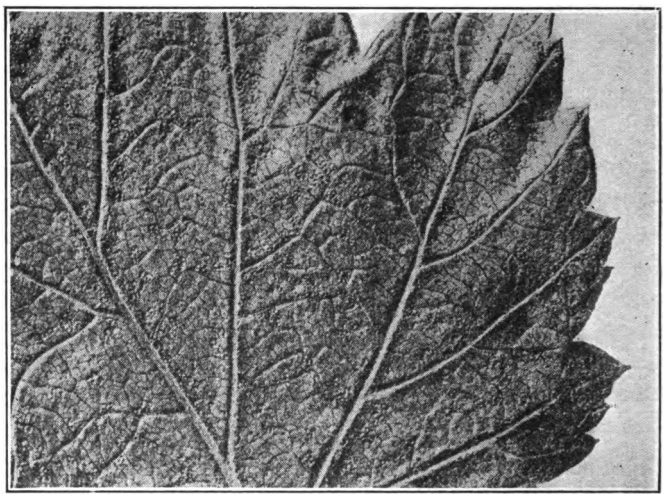

FIG. 55. — European currant-rust; uredinia on lower surface of black currant leaf.

fusiform, or spindle-shaped, swellings which taper upward. These are usually found on wood at least three years old. When a swelling is first noticeable, small transparent spots appear which develop into blister-like pustules surrounded at the base by a translucent spot, whence the name blister-rust. These pustules break open and drops of sweet, honey-colored fluid exude. Later, spore-pustules break through the bark. These are small, scattered, and from them orange-colored masses of spores push forth through the break in the bark (Fig. 57).

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After the spores escape, a whitish membrane is left about the edges of each pustule (Fig. 57); this disappears by the first of June and an empty depression remains. Affected trees are stunted: the tops have a peculiar bunched growth, the past season's growth is shortened, and finally the needles turn yellowish in color.

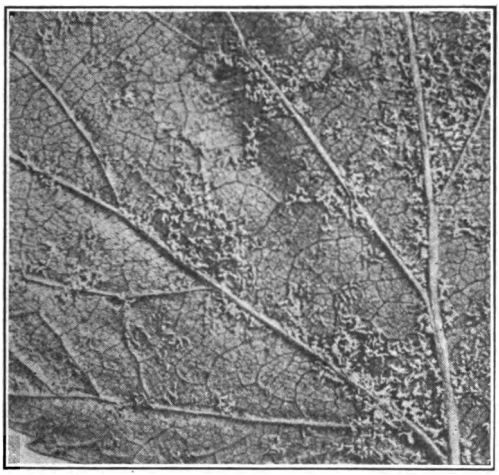

FIG. 56. — European currant-rust; telia on lower surface of black currant leaf.

Cause of European rust.

This rust fungus, *Cronartium Ribicola*, like many others, is heteræcious; that is, it requires two distinct kinds of hosts for its full development. And during its life-history five spore-forms are developed. The fungus attacks and lives in the bark of the white pine in one stage of its life-history; as such it is known to scientists as *Peridermium Strobi*. It may also occur on other five-needle pines, such as the stone-pine (*Pinus Cembra*), sugar-pine (*Pinus Lambertiana*), western white pine (*Pinus monticola*), and Himalayan white pine (*Pinus excelsa*). The white pine (*Pinus Strobus*) is found in North America from Newfoundland to Pennsylvania, along the Appalachians to Georgia, west to eastern Iowa and Minnesota; in Canada from Lake Winnepeg to the northern shore of the St. Lawrence Gulf and Newfoundland. In the other stage, the fungus lives in the leaves of about twenty-five different species of wild and

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cultivated currants and gooseberries; this stage is known as *Cronartium Ribicola*, the name now used to apply to any and all stages of the fungus on whatever host it is found.

The fungus lives from year to year in the bark of living pines (Fig. 57), finally fruiting and developing a crop of æciospores which blow to Ribes (currant and gooseberries) near by. In pines the organism may be shipped thousands of miles. It is not known whether the fungus hibernates on the Ribes or not, although there is some evidence that such is the case. In the spring from early April to June (chiefly in May), the æciospores are blown to young Ribes leaves. These spores are produced over a period of about two weeks. They apparently are never carried more than a few hundred feet. Should any of these spores fall on pines, infection will not result; the fungus cannot pass from pine to pine; it must first go to the currant or gooseberry, if it is to grow. In the presence of moisture the æciospores germinate and infect the Ribes leaves. Cold weather inhibits the rapid progress of the fungus in the currant leaves. With a favorable temperature the fungus soon establishes itself, and after two weeks a new crop of spores is produced. These are always produced on the lower surface of the Ribes leaf. They are distinct from the æciospores which come from the white pine, and are known as uredospores. The uredospores are capable of infecting other currants and gooseberries. This stage is sometimes called the summer or repeating stage (Fig. 55). This repetition may proceed throughout

FIG. 57. — European currant-rust; æcia on white pine.

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the growing-season, a new generation of uredospores being produced every two weeks if conditions favor. Thus the fungus spreads rapidly and over great distances, assuming, of course, that Ribes are present.

From the latter part of July until leaf-fall, spores of another kind are formed on the currant and gooseberry leaves (Fig. 56). They are recognized as groups of three to twelve stout threads, measuring a quarter of an inch or less in length. The spores of this stage are called teliospores. The teliospores differ from the uredospores in that they cannot reinfect the currant and gooseberry. They germinate in a peculiar fashion: a short tube is produced on which small spores, called sporidia, are developed. The sporidia are extremely light and are blown easily by the wind. These blow to the white pines, germinate, and infect the bark of the same. The mycelium resulting spreads through the bark for several inches, but there may be no external evidence of the disease for several years (one to six or more). Finally the bark begins to thicken, and the first external evidence of the fungus itself consists of minute bodies known as pycnia; these appear from June to September. Closely following the development of the pycnia are the æcia. Spores from the æcia blow back to the currants and gooseberries.

Control.

From the point of view of the people at large the removal of the less important host would involve the currant and gooseberry. But in some cases the commercial grower of bush-fruit should destroy the white pine only in those cases where but a very few trees stand in close proximity to the berry plantation. The safety-zone may be put at five hundred feet; that is, the two hosts, white pine on the one hand, and currants and gooseberries on the other, should not be allowed to grow within a distance of less than five hundred feet of each other. It is safer at present to destroy those Ribes plants which are persistently

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diseased; for there is a slight possibility of their carrying the blister-rust fungus over winter.

Considerable effort has been made to date to control the disease with reference to the five-needle pines. So far, however, the work has not been wholly successful. The difficulties involved are these: (1) There has never been any attempt previously, in Europe or America, to eradicate a tree-fungus of this sort; so there is no past experience on which to base the procedure. (2) The fungus incubates, or lies dormant without showing itself externally, for a period varying from one to six or more years; thus inspection has been inefficient. Diseased trees have been overlooked. (3) Neither the federal nor the various state officials who must carry on this inspection have the power to destroy the currants and gooseberries found within the danger zone. (4) The people generally do not realize the seriousness of the blister-rust disease, and therefore cooperation has not been unanimous.

With the knowledge of the disease now available it seems likely that rational measures may be employed even in the absence of previous experience. On account of the long incubation period of the fungus in the pine, annual inspections are necessary, unless, of course, the whole group of pines be destroyed immediately on discovery of the disease. Special emphasis is laid on the destruction of wild currants and gooseberries, for the uredospores may spread from plant to plant for many miles during the summer. There is great need for adequate state laws. Inspectors should be regarded as friends of the people rather than as personal enemies to the fruit-grower or the forester. Their presence should be taken as an index of precaution against calamity rather than of any ill-influence of a supposedly antagonistic citizen. Such officers not only need cooperation, but, unfortunately, as experience has shown, they need legal power to compel concerted action in the eradication of diseased plants. In most cases the removal of currants and

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gooseberries, whether healthy or diseased, is essential. The situation is critical, and unless the proper measures are taken at once the disease will have reached a range beyond any possible control. The fruit-grower must face this problem: that, irrespective of his innocence and his personal feelings in the matter, the total destruction of his currant and gooseberry bushes should not be delayed when he has the official information that such should be done. In a preliminary way the grower can cooperate by making inspections of Ribes, beginning about July first.

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ANGULAR LEAF-SPOT

Caused by *Cercospora angulata* Winter

This leaf-disease of currants is common in Iowa and New York. It is also found in Ohio, Michigan, West Virginia, Maryland, Nebraska, Wisconsin and Rhode Island. In some years it is very destructive.

The spots produced by *Cercospora angulata* are readily recognized. On the upper surface of a leaf the spots are of a paler color than the surrounding healthy tissue. The under

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surface of the diseased portion is smoky in color. The lesions are roundish or angular, and measure from one-sixteenth to one-eighth of an inch in diameter. Little attention has been given to the life-history of the pathogene.

The disease may be controlled by the schedule advised for the Mycosphærella leaf-spot (see page 205).

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American Powdery-Mildew

Caused by *Sphærotheca mors-uvæ* (Schw.) B. and C.

This is believed to be primarily a gooseberry disease both in Europe and in America. It may, however, cause considerable injury to currant bushes, as was noted in July, 1915, on black currants in a New York State nursery. The disease was then found affecting the varieties Champion, Naples and Saunders, while red currants near by were resistant. The trouble has also been reported from Ohio, Nebraska, Washington and California.

The affected leaves and berries are covered with a brownish felt-like growth.

Although the causal fungus, *Sphærotheca mors-uvæ*, is morphologically the same as that causing gooseberry-mildew, yet limited observations indicate that the pathogene on the currant is so specialized that it refuses to infect even the most susceptible varieties of gooseberries. Conversely, the fungus, even when abundant on gooseberries, is rarely found on currants growing in close proximity. (See Gooseberry, page 222.)

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ARMILLARIA ROOT-ROT

Caused by *Armillaria mellea* (Fries) Quel.

Among the many plants affected by the Armillaria root-rot is the currant. Severe injury is reported from the Pacific Northwest. Symptoms, cause and control are discussed under Apple, page 96.

FOMES ROOT-ROT

Caused by *Fomes Ribis* (Schum.) Fr.

This is a root disease which has been observed in New York and Minnesota. It probably occurs elsewhere in the United States, since it affects sassafras as well as currants. The fungus *Fomes Ribis* produces its fruiting bodies around the base of the bush. These are tough, woody, thin, rough, brownish punks, and measure one to two by two to six inches. The diseased roots are abnormally dark, and finally are killed. The destruction of affected plants is advised.

SILVER-LEAF

Caused by *Stereum purpureum* Fries

This disease, which is by far the most common on the plum, is sometimes found on the currant. As yet no considerable injury is reported on the latter host. (See Plum, page 368.)

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CHAPTER VIII

GOOSEBERRY DISEASES

COMPARATIVELY few gooseberry diseases are of a serious nature; they are seldom sufficiently injurious to create anxiety. The American powdery-mildew and the leaf-spot are the most prevalent diseases of this fruit. The gooseberry, like the currant, is affected by the European rust (white pine blister-rust), but is of less importance in this connection than the currant.

AMERICAN POWDERY-MILDEW

Caused by *Sphærotheca mors-uvæ* (Schw.) B. and C.

The American gooseberry mildew is indigenous to the United States, where it is has been known for at least three-quarters of a century. It probably originated in this country on wild gooseberries before this fruit was brought under cultivation. It is believed on good authority that the fungus was carried from the United States to southwestern Russia about 1890, from which point it spread into neighboring European countries. The disease made its first appearance in western Europe in 1900, when it was found in Ireland. Undoubtedly the mildew fungus was introduced from America about that time. In 1901 it broke out again and became increasingly prevalent during the four years succeeding. With Russia and Ireland as centers, the disease soon became prevalent throughout Europe wherever gooseberries were cultivated.

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The disease is far more serious in Europe than in America. It is a well-recognized fact that when a fungus is introduced into a new and favorable climate and among new varieties of hosts, it is liable to become more destructive. In this respect the gooseberry-mildew pathogene is no exception. It is now established in the Old World and has become a permanent factor in gooseberry culture. While the fungus is less virulent in America, yet it is one of the most serious enemies of this fruit. The berries are usually rendered unsalable, and sometimes are even destroyed. The vitality of affected bushes is reduced, and they are more susceptible to winter-injury than those which remain free from mildew.

Symptoms.

The lower parts are first affected. The disease appears in May or June. In severe cases not a single berry remains free from the attacks of the pathogene. The leaves and young canes are also affected. At first there is a superficial whitish growth having a moldy, powdery appearance. Several spots may coalesce to form large patches. The leaves and tips of the stems may become distorted in serious cases. Affected berries are sometimes dwarfed. Finally the fruit may crack and decay. In later stages the mildew patches become buff or rusty-brown in color. Scattered through the felty growth, small black specks may be observed.

Cause.

The American gooseberry-mildew fungus is known as *Sphærotheca mors-uvæ*. The small specks mentioned above are perithecia of the parasite. Within each of these bodies is found a single ascus containing eight ascospores. These spores remain in the perithecium during the winter, either on the fallen leaves or on the canes of the past season's growth. Sometimes, when the leaves disintegrate, the perithecia hibernate in or on the soil. In the spring when the leaves are half grown, or later, the ascospores are forcibly ejected from the

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perithecia. The spores come to rest on the young leaves or fruits. Following germination of the ascospores a weft of mycelium develops which grows superficially. At certain places, however, small sucker-like bodies, called haustoria, are sent into the host cells; these are the organs of feeding. Nourishment is derived from the host and the mycelium grows. In the early part of the growing-season summer spores are formed. They are produced in chains on erect branches sent up from the weft of surface mycelium. The conidia (spores) are produced in great numbers, and soon fall away, giving the lesion a dusty or powdery aspect, whence the name powdery-mildew. The conidia are scattered by the wind, and they give rise to new infections similar to those just described. Toward autumn the perithecia are formed, and these serve to carry the fungus through the winter.

Control.

This is one of the most difficult of powdery-mildews to control, particularly in wet seasons. However, if remedial measures are thoroughly applied, less difficulty will be experienced. The work may be done at a slight expense.

The standard remedy has been to spray with potassium-sulfide (liver-of-sulfur), one ounce to two or three gallons of water. In New York State and elsewhere lime-sulfur solution, at the rate of one to forty, has proven effective. In Oregon it has been found that an application of lime-sulfur at dormant strength on dormant branches, followed by a solution diluted one to thirty, gives excellent satisfaction. It is suggested that it may be desirable to use potassium-sulfide in later applications, since lime-sulfur leaves a deposit on the sprayed parts. The applications of a fungicide should be made as follows: (1) when buds break open; (2) at intervals of ten days until at least five applications have been made. The more susceptible varieties may demand more sprayings.

Pruning in the fall may reduce the amount of the inoculum,

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although there are few experimental data on which to base positive recommendations. The rapidity of the spread of the fungus in Europe has necessitated legislation concerning it. Considerable money has already been spent along this line in England, but the disease is still serious in spite of all efforts. It is advisable that weeds be kept from gooseberry plantations, especially beneath bushes in order to allow free circulation of air.

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Leaf-Spot

Caused by *Mycosphærella Grossulariæ* (Fr.) Lind.

This leaf-spot disease of gooseberry (Fig. 58) is the same as that found on the currant. It prevails to some extent in America wherever the host is grown, having been reported from at least ten states over the northern half of the United States. It is probably most important in Ohio, New York and Iowa, where defoliation of affected gooseberries may be more extensive than of the currant. This may take place before maturation of the fruit, in which case the berries wither and are rendered worthless. Native gooseberries are injured more than other varieties; this is true in New York at least.

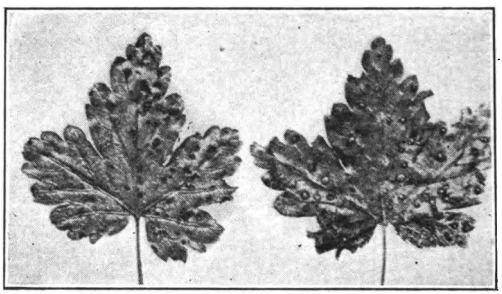

Fig. 58. — Gooseberry leaf-spot.

The control of this trouble is said to be accomplished with more ease than in the case of the same disease on currants. For the schedule of remedial measures, see Currant, page 205.

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(See references under Currant, page 205.)

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European Currant-Rust

Caused by *Cronartium Ribicola* Fisch. von Wald.

The European currant-rust, or white pine blister-rust, is known to occur on the gooseberry, although it is notably less common on this plant than on the currant. The characteristics of the disease on the gooseberry may be understood by reading the account as presented under Currant, page 212.

Cluster-Cup Rust

Caused by *Puccinia Pringsheimiana* Kleb.

The cluster-cup rust is exceedingly common on wild gooseberries and currants and is known to some extent on cultivated forms. It occurs in Europe, Alaska and in many parts of the United States. Serious damage is seldom reported, although some complaint was made several years ago (1892) of the loss of foliage and fruit on account of an unusual outbreak.

Symptoms.

The disease may be recognized in the early summer by the presence of bright, orange-colored cups grouped on reddish, swollen areas on the lower surface of the leaf. Sometimes these structures are found on the fruit, causing distortions.

Cause.

The cluster-cup rust fungus is *Puccinia Pringsheimiana*. The orange-colored cups already described contain spores known as æciospores. These are carried to various common sedges, *Carex acuta* and *C. Goodenovii*, where infection takes place. Following the development of the mycelium within the sedge leaves, a second type of spore structure, known as a uredineum, is produced; the uredinia bear uredospores. Later a third kind of fruiting body appears; this is known as a telium, and it bears teliospores. In the latter condition the winter is

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passed. In the spring the teliospores germinate by means of a short promycelium bearing sporidia. The sporidia are blown to the currant and gooseberry, where infection occurs. Finally, the æcial cups appear; these bear a fringed margin and are filled with small æciospores.

Control.

Usually this rust disease does not cause damage sufficient to warrant particular attention. If it should assume serious proportions, the elimination of rusted sedges in the fall would be essential. No further control measures are known.

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Armillaria Root-Rot

Caused by *Armillaria mellea* (Fries) Quel.

The gooseberry, among several other fruits, is at times badly injured or killed by *Armillaria mellea* in the state of Washington. The disease probably occurs elsewhere on the gooseberry, although it has not been so reported. Its importance on tree-fruits gives it a prominent place in the realm of plant diseases. The symptoms, cause and control as discussed under Apple (page 96) are applicable for the most part to the gooseberry.

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CHAPTER IX

GRAPE DISEASES

In most regions of grape-culture, the vine is as much subject to destructive diseases as any other of the less important fruits. But on the Pacific slope vines are notably free from fungous troubles, owing to the light rainfall of the summers. This dependence of disease-producing organisms on weather conditions is also exhibited by fungi on other crops, so that the freedom from grape-disease in the region mentioned is not peculiar.

The serious attention of American plant pathologists was not given to grape diseases prior to 1887. In earlier days these troubles were so detrimental to grape-production that efforts to grow this fruit, almost without exception, were unsuccessful.

Grape-culturists, in America and Europe alike, are prone to censure each other for their imported troubles. But it should be remembered that while the powdery-mildew and the anthracnose fungi were being sent to us by Europeans, the black-rot and downy-mildew pathogenes were carried from the United States to European vineyards. A knowledge of these historical facts has a practical application in the control-program. For it is now well-known that native fruit-varieties are more resistant to indigenous fungi than to introduced fungi. European stock may therefore be satisfactorily grown in conditions favorable to introduced fungi, like those causing the powdery-mildew and anthracnose, but such stock is highly susceptible to black-rot and downy-mildew. Similarly, it may be expected that varieties native with us will succumb less readily under

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the attacks of the black-rot and downy-mildew pathogenes than when besieged with powdery-mildew and anthracnose fungi. From these facts it is clear that the pedigree of a variety is an essential indication of the probable susceptibility to disease.

Black-Rot

Caused by *Guignardia Bidwellii* (Ellis) Viala and Ravaz

The first important records of black-rot come from Missouri in 1861, although the disease had been known many years prior to this date. About that time (1861) there was a rise in the grape industry near St. Louis, which, especially in 1860 to 1864, was accompanied by epiphytotics of black-rot. In 1885 the disease was first recognized in France, into which country the pathogene had, at some previous date, been introduced from America. In 1886 there was very little of the disease, owing to the dry weather; however, in 1887 it became serious in many localities new to the trouble. The vineyardists abroad were desirous of obtaining the best varieties and so they went not only to the wild grapes for cuttings, but imported them from America. They were further interested in getting possession of varieties resistant to the phylloxera, and consequently the introduction of the black-rot organism was comparatively easy. So serious had the grape-disease situation become in France by 1887, that the noted Viala was detailed by the French government to visit America in the interest of French viticulture. Since this date many papers dealing with the disease have appeared in various languages, but more particularly in French and English.

At present the black-rot disease is practically omnipresent. Its geographical range in Europe is approximately coincident with that of the grape; for the pathogene, having reached France, spread rapidly into Germany, Italy and Asia Minor.

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In the United States it may be found in all grape-growing regions except in the state of California, where the conditions are highly unfavorable to it. In that state it is almost unknown, but its place is taken by the powdery-mildew fungus.

Wherever found, this disease is of great importance because of the injury it produces to all of the parts above ground. There may result serious loss by the reduction in the amount of fruit; and the leaves may be spotted to such an extent that they die. The very general range of the trouble makes it a constant foe, especially wherever susceptible varieties occur. All authorities are agreed that black-rot is the most serious fungous disease with which grape-growers generally have to contend. The amount of loss occurring each year varies with the season, there being an abundance of it in wet seasons and much less in dry ones. The disease is more troublesome in warm, humid regions than cool, dry ones. In North Carolina the heavy losses come within a week after blossoming-time, while in New York the greatest injury is incurred when the berry is one-half to two-thirds grown. In many sections of the country the destructive nature of this disease has been responsible for the abandonment of grape-growing. In 1906 Michigan vineyardists lost 30 to 40 per cent of their crops on account of black-rot; in Ohio in 1905 the loss is put at 30 per cent of the crop, meaning a loss of $28,500. The losses in many regions are practically total; this has been true in several states. A case is on record for 1906 where a large vineyard of 200 acres in New York did not yield enough grapes to pay for operating-expenses; the reduced yield here was due to the action of the black-rot pathogene.

Symptoms.

As a rule all the green parts of the vine may be affected with black-rot. The old portions and the fruit of *rotundifolia* varieties, like the Scuppernong, in southern United States are rarely attacked.

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The name black-rot, which is in use in America and Europe, applies to the condition of affected grapes (Fig. 59). When the berries are about one-half grown, the disease shows its first symptoms in the form of a blanching. This is soon replaced by a whitening of the diseased area, which is now more evident. A brownish line appears at the margin, and there results, on account of a light-colored circular disk with an encircling darker band, a bird's-eye effect. Some grape-culturists confuse this stage with the true bird's-eye, or anthracnose, described on page 250. The spot increases in size rapidly and its surface becomes sunken. In a few hours after the beginning of the development of the lesion, numerous minute brown specks appear on the light-colored center. These are the fruiting bodies of the black-rot organism, which very shortly become so numerous as to give the spot a blackish aspect. Occasionally the extent of the lesion is halted, and in a few days there is formed a thin black superficial crust on the side of the berry. The usual course of development, however, is for the berry to become entirely in-

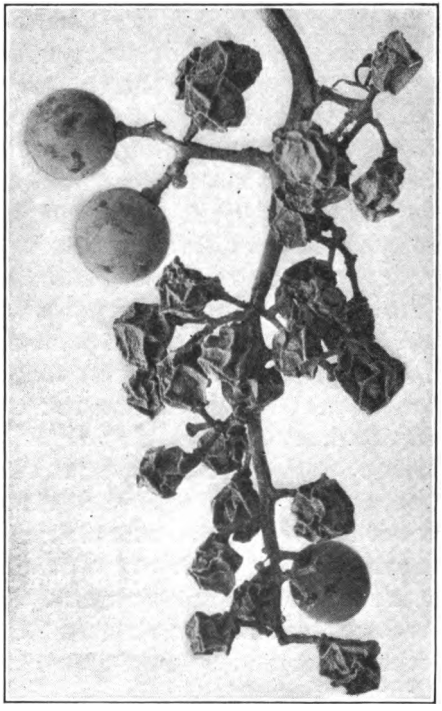

FIG. 59. — Black-rot on grape berries.

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volved. After about a week the berry is a hard and shriveled mummy (Fig. 59).

Although the berries, of all susceptible parts of the vine, are most severely affected, the disease may first be observed on the leaves (Fig. 60). In June or early in July black-rot appears on the leaves in the form of reddish brown, somewhat circular spots. Close observations show that the first evidence of the trouble on the leaves is a slight blanching of the affected tissue. The small veinlets form the margin of the spot so that, while the shape of the lesion tends to be circular, the outline is finely crenulate (Fig. 60). Soon the center of the affected area becomes ashen-gray, but at this stage the margin is still brown. By the time the spot is just visible to the unaided eye the margin appears as a black line. Two or more spots may coalesce to form large irregular areas. The fruiting bodies of the pathogene protrude through the cuticle of the leaf. These structures are black and are arranged promiscuously or in more or less concentric rings (Fig. 60).

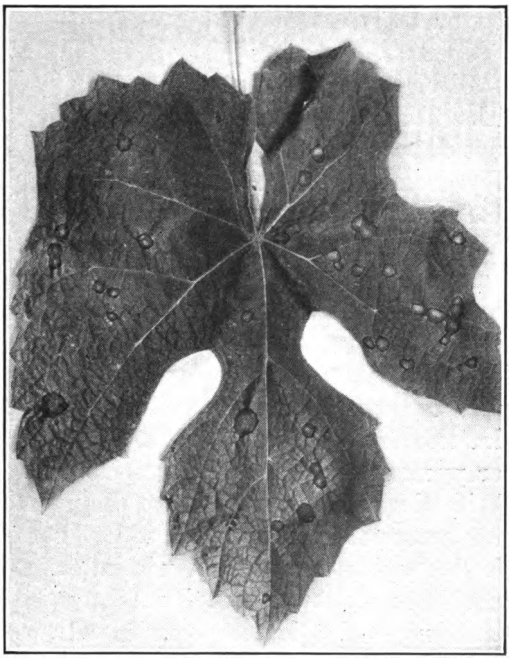

FIG. 60. — Black-rot lesions on grape-leaf.

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The black-rot lesions occur as small dark depressions or cankers on the stems, tendrils, peduncles, petioles and leaf-veins. On the canes the spots rarely extend more than a quarter of the distance around, but the tendrils and leaf-petioles may be nearly or wholly girdled. The affected areas are small, one-twelfth of an inch to one inch long, and are elliptical or considerably elongated in form.

Cause.

Black-rot is produced by the fungus *Guignardia Bidwellii*. The trouble begins in the spring, when both ascospores and pycnospores are liberated from their hibernating quarters. The former spores are discharged with considerable force into the air for some distance. Subsequently they are carried by air currents to a variety of places. Some of the spores perchance fall on the fruits, leaves or shoots, where black-rot lesions may ultimately be produced. The pycnospores ooze out of the pycnidia which have hibernated on old canes, tendrils, leaf-petioles or mummies. They are washed by rains to the above-ground susceptible parts, where they may finally induce black-rot areas. Either of these spores — ascospores or pycnospores — then, may start the trouble in the spring. With the presence of moisture they germinate and the resulting germtubes penetrate the vine. For some time the fungus is developing a mycelium within the attacked organ, and it may be many days before there is any visible evidence of disease. The length of this period varies with the weather conditions, and with the part affected. When it is hot and dry the period is materially reduced, and with cool weather it is lengthened. In tender, juicy fruits this period is shorter than in stems and leaves. On fruits the period is from 8 to 18 days; on leaves, 10 to 21 days.

The diseased berries show mycelium both between and within their cells. Eventually the cells are killed, they collapse and the surface becomes sunken. Similar action takes place

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in the leaves, canes and other attacked organs. Soon there follows the development of fruit-bodies, the pycnidia. The mycelium forms a gnarl just beneath the epidermis, and in a few hours a crop of pycnospores is matured. These are imbedded in a mass of gelatinous matter, and when moistened a marked swelling of the mass occurs and the pycnospores are forced out in a coil. They are subsequently scattered to other leaves, fruits and woody parts, where new lesions are developed. This process is repeated many times through the summer. With the advent of autumn the fungus ceases vegetative activities and prepares for winter. Some of the pycnidia live over until spring, as already stated. In other cases, generally in August, sporeless pycnidia appear on mummies; in place of spores the cavity of this body is filled with a whitish cellular tissue. In such a state the structure is sometimes referred to as a pycnosclerotium, and as such the fungus passes the winter. In the spring the solid interior of the pycnosclerotium is replaced by asci which eject most of their spores in June. Some of the ascospores are not discharged until later, and may even be found as late as October. With two kinds of spores always present in a vineyard of affected plants it is not difficult to understand how unsprayed, susceptible varieties may be ruined in a favorable season of considerable rain.

Control.

Black-rot has long been kept in check by the application of bordeaux mixture. It is essential to successful protection of the vines that the spraying should be made before rains and that the work be thoroughly done. Apply bordeaux mixture 5–5–50 as follows: (1) when the second or third leaf is showing; (2) before the blossoms open; (3) after the blossoms have fallen; (4) about 10 to 14 days later; (5) again in 10 to 14 days.

In spraying and in the employment of other measures against black-rot the following matters should be kept clearly in mind:

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(1) The disease is of long-standing the world over and is well known from experience to be destructive. In regions where it occurs practically every year to a greater or less extent, the application of the above scheduled sprayings should not be neglected.

(2) All parts above the ground are likely to be affected. Spray every part accordingly. Watch for the disease in June.

(3) The fruit is rotted, the leaves are spotted, and the woody parts are cankered as a result of the action of the mycelium of *G. Bidwellii.*

(4) The inoculum comes from (*a*) old fallen mummies, (*b*) clingers, (*c*) tendrils and canes. Get rid of these sources of trouble as far as possible. Destroy mummies by carrying them out at picking time. Plow the vineyard in the spring: this is commendable both as good viticultural practice and in order to bury fallen mummies. It is not believed a profitable practice to collect and burn clingers. Nor are sanitary measures especially applicable to cankered tendrils and canes. The grower must depend upon bordeaux mixture for the protection of these parts.

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Downy-Mildew

Caused by *Plasmopara Viticola* (B. and C.) Berl. and de Toni

This disease was first observed in America in 1834. Some time prior to 1878 the pathogene was carried to Europe on American stock, for in September of that year it was first recorded in France. During the five preceding years, French growers had been warned against such importations from the United States on account of the downy-mildew of grapes. At that time European vineyardists were eager to use the American root stock on which to graft French vines as a remedy for the grape phylloxera, another pest of American origin which had already been so destructive to French viticulture. But in spite of the admonitions of the French scientists against importations from America, the downy-mildew finally reached the European vineyards. From France the mildew-pathogene spread throughout Europe, where it is now a very troublesome foe. In the United States the disease is very generally known, being more destructive to European varieties than to domestic forms. No variety is notably resistant under all conditions. Both smooth and pubescent, wild and cultivated, sorts are affected. The downy-mildew also occurs on five-leaved ivy, a close relative of the grape.

The greatest losses to American viticulturists from this disease are incurred in northern United States, where in some localities it has been estimated that 25 to 75 per cent of the crop is destroyed. Losses in Europe have been enormous. The disease is destructive not only to the berries but to practically all young or green portions of the vine. The nature of the losses is as follows: (1) shelling; rotting and mummification of the fruit; (2) spotting of the foliage, often resulting in premature defoliation; and (3) sometimes a dwarfing and killing of canes and leaves.

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Symptoms.

On the upper surface of the leaf the first signs of downy-mildew are in the form of small greenish yellow indefinite spots, the margins of which gradually merge into the darker green of

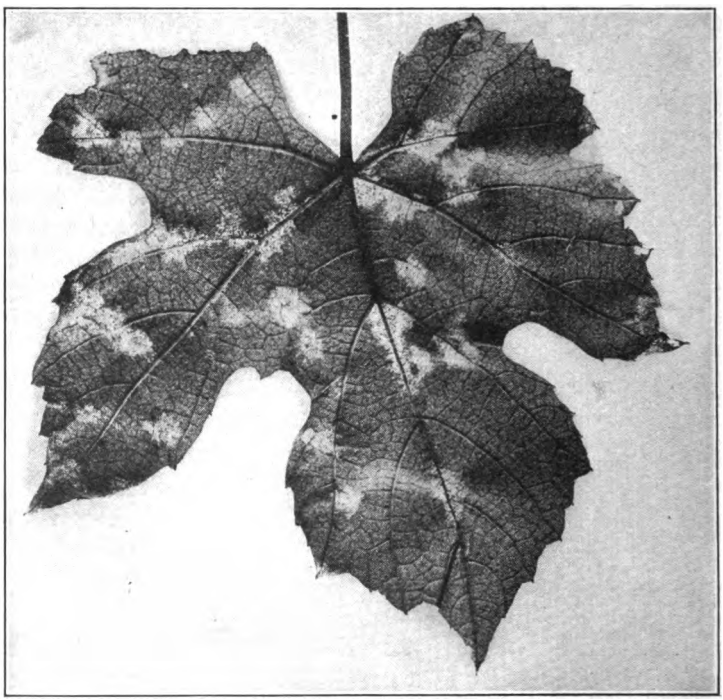

FIG. 61. — Downy-mildew on lower surface of grape-leaf.

the leaf. In a short time there appears within the spot a network of small, reddish brown lines, the discolored smaller veins. These lines become more pronounced until the whole diseased portion is brown, dry and brittle, and eventually cracked. These symptoms apply particularly to susceptible leaves. On the foliage of the more resistant vines there occurs

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only a slight change in color; within this area are found numerous brown punctations, which do not unite to form a dead, brown spot in the leaf-tissue. The disease on the lower surface of the leaf is at first similar to that on the upper, but is very soon covered by a downy, white growth (Fig. 61) which is most pronounced just beneath the greenish yellow portion mentioned above. On the brown portion the whitish felt, the mildew-pathogene, soon disappears. The amount of mildew on a leaf depends on weather conditions and on the variety. The lower portion of a vine may be denuded of its foliage.

The canes, leaf-petioles and tendrils are subject to attack, especially in wild varieties. At first the affected portion has a water-soaked, shiny appearance, which is attended by a local swelling of the tissue. The lesion is bare at first, but soon the mildew covers it. In older cases the affected area becomes brown and dead, and a depression results. In cases of severe attack the cane is dwarfed, the leaves remain small, and the cane may die (Fig. 62).

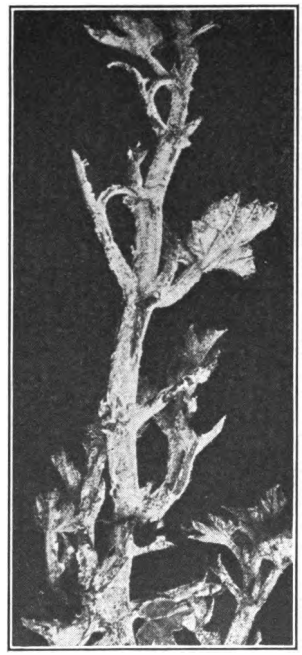

FIG. 62. — Downy-mildew on grape-shoot.

The fruit (Fig. 63) and flowers of wild varieties commonly show the downy-mildew. The attack may come at any time from flowering to maturation. The first sign of the trouble on the fruit is a hardening of the berry, together with a change from its normal color to a grayish blue lead-color. It is during this stage that the mildew appears. In later stages the berry

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withers, turns brown or red, and finally shrivels into a mummy (Fig. 63). Diseased fruits shell very easily.

Cause.

The downy-mildew pathogene is a fungus, *Plasmopara Viticola*. It has both a sexual and an asexual stage, the former

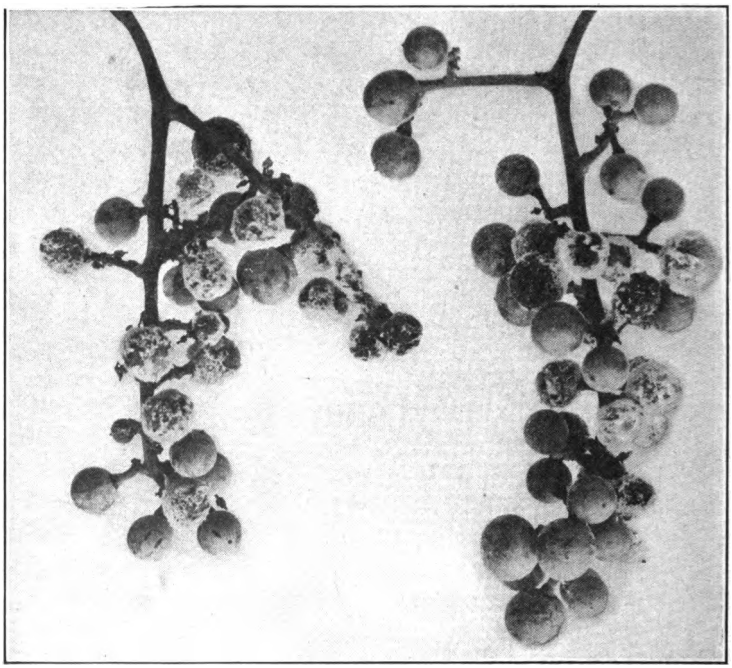

FIG. 63. — Downy-mildew on grape-berries.

represented by oospores, the latter by conidia. It is generally agreed that the oospores carry the fungus through the winter in the old fallen leaves. In the spring these oospores germinate, forming a conidiophore, on which is borne another spore, a conidium. The conidium in turn, at maturity, germinates, but instead of forming a germtube, its contents break up into

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six or eight extremely small naked spores. Each of these has two cilia which give it the power of motion; on account of this motile character these small naked bodies have been named swarmspores. The production of swarmspores, which really initiate the disease, begins in February and continues until June. They are formed and discharged within 24 hours. The swarmspores or their parent spores, the conidia, are spattered in drops of water or mud by heavy rains to the leaves of the vine. After reaching the leaf, the swarmspore, which is still a naked protoplasmic mass, soon comes to rest and surrounds itself with a thin wall. Then a germtube is developed which penetrates the leaf by way of a stoma, always on the lower side of the leaf. If a swarmspore is on the upper surface of the leaf, it swims over the edge of the same to the lower side. The germtube within the tissues develops into mycelium which brings about the various changes in the affected parts as evidenced by the symptoms already described. Within a short time the fungus masses itself beneath a stoma, through which opening several (four to ten) conidiophores pass. These grow from most of the stomata in the infected region, each conidiophore reaching a height of about one-fiftieth of an inch. In mass they present a whitish, downy appearance — the downy-mildew. These structures form branches, on the ultimate tips of which are borne conidia. The conidia are blown to other susceptible parts of neighboring vines, where new spots are formed. In the autumn, affected leaves fall to the ground, carrying the fungus within them. Before winter arrives oospores, as many as 200 to the square centimeter, are formed. In the spring the oospores are set free by a rotting of the leaves, germination occurring in the soil.

A temperature of 77° to 82.4° Fahr. is best for the growth of the fungus. Shaded, moist situations are favorable for its development. Long, dry periods do not kill the pathogene, although such conditions seriously check it. Heavy rains

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of short duration followed by sunshine and winds are not favorable to the spread of the fungus. Most injury is done during hot, wet weather.

Control.

Since the fungus hibernates in old fallen leaves it is commonly recommended that these be plowed under early in the spring. The real value of such an operation, however, is not definitely known. The vines should be sprayed to protect them against attacks of the downy-mildew pathogene in any case. Use bordeaux mixture 5–5–50, making five or six applications during the season. The first should be made just before the blossom buds open; the others at intervals of two weeks.

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Powdery-Mildew

Caused by *Uncinula necator* (Schw.) Burr.

The powdery-mildew, or oidium, of the vine is native to the Old World, originating on native plants in Japan. It was at one time erroneously held that this disease, like black-rot and downy-mildew, originated on wild vines of the eastern and central United States. The disease first appeared in Europe

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in 1845, when it was found in England. Two years later it reached France, and in 1848 was first observed in Belgium. By 1850 great devastation was wrought in the vineyards of France, and that same year it appeared for the first time in Spain and Italy. The following year powdery-mildew became general over France and was then (1851) found in Hungary, Greece, Switzerland, Syria, Asia Minor and Algeria. Great damage was caused by the disease in 1854 in France. After that date growers began to learn how to control the trouble as a result of the discovery and perfection of the use of sulfur. By 1859 the use of sulfur had become so general that the crop of grapes had returned to its normal size. In 1866 the disease made its appearance in Australia. In the United States it has a wide geographical range, occurring from Massachusetts to Florida and westward across the continent of North America to the Pacific Coast. The first record of the oidium in America comes from California; it was known in that state as early as 1859, but doubtless occurred there long before that time. The disease is also found to some extent in Canada, particularly in Ontario.

The losses from powdery-mildew of the vine are greatest in Europe. Soon after the pathogene was introduced into Europe losses of ninety to ninety-five per cent occurred in French vineyards, and the damage was so great that in certain regions vineyardists emigrated. In all countries where the disease was then known government commissions were appointed to investigate the matter, and consular reports were issued. Enormous losses were incurred in 1854 and 1855, in which latter year the climax was reached. The disease still occasions considerable annual loss. In certain regions of America, for example in the Chautauqua grape belt of western New York, in California and Oregon, powdery-mildew ranks ahead of downy-mildew, black-rot and anthracnose. This is perhaps due to the ability of the causal fungus to withstand the dry atmosphere

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better than the fungi concerned in the other diseases mentioned. If not controlled, the oidium does considerable damage in western vineyards. In the Chautauqua region of New York growers may in cases of severe infestation lose fifty per cent of their grapes on account of shelling due to the powdery-mildew fungus. The Lindley is very susceptible to the disease.

Symptoms.

All green parts of the vine may be affected: leaves, canes, flowers and fruit. The young leaves at first show whitish or greenish white patches on the upper or lower surfaces. A mottled appearance is thus produced. These patches later run together until a large portion of the leaf is covered with a grayish white mildew. Sometimes this dense growth of the pathogene is accompanied by malformation and discoloration of the affected leaf. A vine with diseased foliage has a wilted and dwarfed aspect; this is particularly true in the earlier parts of the season and in warm, dry weather. An affected vine emits a moldy odor. Eventually black, pimple-like fruit-bodies of the fungus are seen scattered over the mildewed area.

Often the mildew is abundant on the shoots. It appears near the base of the canes and at first is not noticeable; later the affected portion acquires a grayish tint and it is then more easily seen. In severe cases whole canes are covered; in mild attacks the mildew is confined to patches. Later affected canes turn dark, owing to the injury to the outer bark-cells. If young canes are affected, they fail to mature properly and often they become blackened over their whole surface; this appearance is sometimes mistaken for anthracnose.

Sometimes the mildew appears on the flowers, if conditions are favorable in the vineyard during blossoming. This occurs regularly in France, but does not occur in eastern United States. Affected blossoms fail to set or develop in an aborted fashion.

Diseased young berries are dwarfed and are caused to drop. If not affected until older, the fruit continues to develop, but

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in an irregular fashion. As described for the canes the outer cells are injured, which accounts for a halting in the growth of affected portions. Sometimes this irregularity of development results in a cracking of the fruit, in which case the berries either become entirely dry and never ripen, or are greatly reduced in size and quality. The fruits are not attacked by the pathogene after they enter the ripening-period. On older, but still immature, berries brown spots appear; subsequently mildew becomes perceptible. The peduncles and pedicels also show the mildew. Very commonly on these parts the pathogene makes profuse growth. By harvest time the peduncles are dwarfed and withered. The development of the grayish mildew is followed by the appearance of black perithecial bodies as described for the other susceptible organs.

Cause of powdery-mildew.

Sexual fruit-bodies, perithecia, of the causal fungus, *Uncinula necator*, probably remain on the canes and leaves or in the soil until the return of summer. Within each perithecium twenty-five to fifty ascospores are found. These are liberated from their asci, and are carried to the growing shoots and leaves. The ascospores are not all ejected at the same time; some may not be discharged until a year later. It is thought that in many cases the fungus hibernates by means of the asexual (conidial) stage; this opinion is based largely on the scarcity of the perithecia in certain regions.

Whether the fungus begins its spring activities by means of ascospores or conidia, the result is a mildewed spot. The whitish mildew is composed of mycelium and conidiophores. When a spore falls on a leaf it germinates, and a copious development of mycelium results. Haustoria are sent into the epidermal cells for nourishment. As a result of this action the cells turn brown, as described under *Symptoms*. From the mycelium elongated, erect conidiophores extend into the air; each bears a chain of conidia. The conidia are light and are easily dis-

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seminated. They have been found as late as the month of October.

In the late summer and autumn perithecia begin to appear among the hyphæ. At first they are yellowish, but they soon turn black. They are numerous, more than one hundred thousand having been counted on the upper surface of a single leaf. Their further history has been discussed. In certain parts of California they are found in June. Their formation seems to depend upon a peculiar set of weather conditions: if the temperature suddenly falls to about 50° Fahr. just after a period of warm, moist weather, they are produced in great numbers. In other parts of the country where these conditions do not prevail in early summer they are more scarce.

The fungus is favored by sheltered, shaded conditions. Thus vines in the open are less likely to show powdery-mildew. While the fungus requires less moisture than most fungi, it will not grow in an exceptionally dry atmosphere. This is noticeable in California, mildew being far more abundant along the coast than in the drier regions. Vines in low places or along streams are often diseased, whereas the remainder of the vineyard is free from powdery-mildew. Rains or fogs, in the spring or early summer, accompanied by warm weather are highly favorable to the organism. It grows between 50° and 95° Fahr., but does so rather slowly except between 75° and 90° Fahr. It ceases growth below 50° and above 100° Fahr. These characteristics of the fungus result in its somewhat erratic appearance in different seasons and in different localities.

Control.

In handling powdery-mildew the following points should be taken into account:

(1) The causal fungus may possibly live for one to one and one-half years in the soil. If the perithecia are buried, they are only preserved until plowed up again a year later.

(2) The fungus is favored by moisture in spite of the fact that

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it can withstand more drought than other grape fungi. Hence anything which permits sun and air to reach all parts of the vine may lessen the danger from mildew: (*a*) remove rows of trees which shade the vines on the south side; it is assumed that this will be done only in the case of trees of small worth; (*b*) plant vines at a reasonable distance apart so that they may dry off more quickly after dews, fogs and rains; (*c*) rows which extend north and south are said to evaporate their moisture most easily; this should be borne in mind when planting grapes in mildew-regions; (*d*) likewise, low-trellised vines are more easily dried by the sun and wind; (*e*) wet places should be drained; (*f*) vines should be pruned so that they will spread; this is of value in the moisture consideration.

The fungus may be eradicated from the vines by the use of sulfur dust (powdered sulfur, or flowers of sulfur); in some regions this may be done cheaply and effectively. Old and young vines alike should be dusted. The sulfur acts by killing the mycelium of the fungus which, it will be remembered, is superficial. Fortunately the sulfur acts at temperatures which are optimum for the fungus, the rate of the killing increasing as the temperature rises from 75° to 100° Fahr. So far as possible, dusting should be avoided when the vines are very wet. Applications of dust to the soil have been shown to have practically no effect on powdery-mildew. In California injury from the use of sulfur may be expected only when the temperature is above 110° Fahr. and only on varieties like the Isabella, Othello and other American varieties. In the Chautauqua belt of New York sulfur dust causes considerable injury and should not be used except on vines of European origin. It has been noticed that sulfur-dusted grapes show less tendency to drop their blossoms, and also ripen their fruit seven to ten days earlier than vines not so treated.

One to six applications of sulfur are necessary, depending on the locality, weather, variety and exposure. Apply as follows:

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(1) when the shoots are about six or eight inches long; (2) during or just before blossoming; (3) later applications are necessary only on very susceptible varieties or where irrigation is practiced when grapes are half-grown. Winter treatments are not generally commendable. The cost, including material and labor, varies from forty to fifty cents the acre for one treatment. Where it is necessary to treat vines for downy-mildew and black-rot, bordeaux mixture may be suitable for powdery-mildew. Liquid fungicides, however, are not as effective as sulfur dust, and in vineyards where this disease is particularly troublesome along with other diseases, it is best to follow the bordeaux sprayings with dust.

The fungus shows preference for European vines; all American varieties are less susceptible. These points are worth remembering in planting the vineyard where powdery-mildew is prevalent.

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ANTHRACNOSE

Caused by *Glœosporium ampelophagum* Sacc.

Grape-anthracnose, or bird's-eye-rot, is widely distributed east of the Rocky Mountains, where at times it is of considerable importance. It was first discovered in central Illinois about 1881 and later was found in many other parts of that state. By 1885 the disease had been noted generally over the eastern and middle-western states. The disease originated in Europe, where it has occurred to a serious extent for many years. The anthracnose fungus was doubtless imported from Europe at some time prior to 1881.

Fortunately the disease does not appreciably affect the Concord, the most extensively-grown of American grape-varieties. Many other favorite varieties, however, are very susceptible to the trouble, especially the Moore's Diamond, Catawba, Salem, Niagara, Diogenes, Brighton, Missouri Reisling, Clinton, Vergennes, Pocklington, Norton, Champion, Thompson's Seedless, Malaga, Tokay and Black Hamburg. On these varieties anthracnose is of considerable economic importance, due in part perhaps to the fact that the disease is not readily controlled. In serious outbreaks the fruit may be almost wholly destroyed, and the vines seriously damaged. The growing of certain desirable varieties in some localities is unprofitable on account of the unusual damage done by anthracnose. It ranks fourth in importance among fungous diseases of the grape east of the Rocky Mountains, black-rot, downy-mildew and powdery-mildew being more troublesome in the order listed.

Symptoms.

All green parts of the vine are subject to anthracnose throughout the growing-season. It is most common, however, on the shoots (Fig. 64) and berries (Fig. 65).

On the shoots and tendrils small cankers are produced (Fig. 64). The lesion is brown, slightly depressed in the center, but

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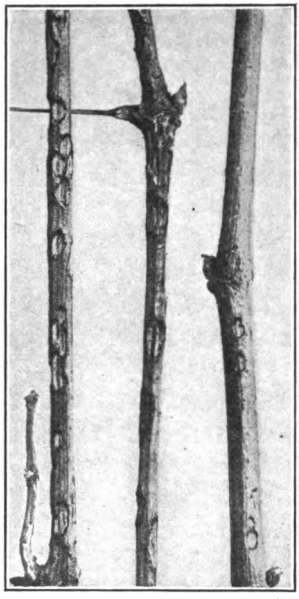

FIG. 64. — Grape-anthracnose on Lindley canes.

raised at the border. The spot enlarges, becoming elongate in the direction of the main axis of the shoot. In later stages the center becomes more depressed, and the color turns grayish.

The bark is finally destroyed, and in severe cases the underlying wood appears burned. Canes are not often girdled, although affected stems bearing clusters suffer in this manner.

On the berries the well-known bird's-eye-spots are produced (Fig. 65). The lesions first appear as small, dark-brown areas; later the color is grayish in the center wherever the cuticle is ruptured, but the border remains dark. The spots increase in size, but instead of elongating as they do on the canes, they remain somewhat circular. Between the gray center and the dark border is a well-defined band of bright red. The appearance thus resulting has given rise to the name bird's-eye-rot. Finally the berries wither and dry up, leaving a mummy. Anthracnose mummies are not as extensively wrinkled as black-rot mummies, and in the former the outline of the originally affected area may still be seen.

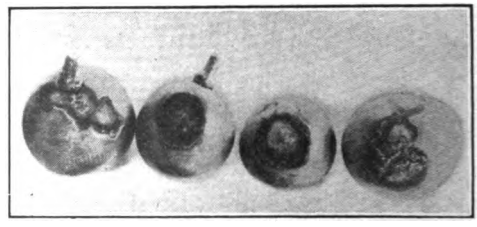

FIG. 65. — Grape-anthracnose on berries.

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In the event that half-grown berries are attacked they become irregular in shape.

The leaves and other parts of the vine when affected by anthracnose show lesions similar to those already described on the shoots. Spots on the foliage are pale-gray with dark-red borders. Cankers are produced on the petioles and veins.

Cause of anthracnose.

The lesions described above are due to the fungus *Glæosporium ampelophagum* (=*Sphaceloma ampelinum*). In America only one spore-stage is known; in France, plant pathologists claim to have discovered a winter sexual spore-stage. Accordingly, the name *Manginia ampelinum* has been substituted. The similarity of the grape-anthracnose fungus to the blackberry and raspberry-anthracnose fungus has resulted in one case, at least, in the suggestion of the possible identity of the two organisms.

The mycelium of the fungus grows within the affected tissue. Certain threads come to the surface and form fruiting bodies called acervuli. These structures burst through the skin of berries and canes, sending out many erect conidiophores on which conidia are produced. These spores ooze out in a gelatinous mass held together by their sticky coatings. The sticky substance dissolves in water and the spores are thus liberated. They are disseminated by rain and dew, the process occurring from the time the berry clusters are yet in the bud until the close of the season. Spores falling on green parts of the vine germinate, and after about a week signs of the anthracnose disease begin to appear. The germ-tubes penetrate the healthy, unbroken epidermis. With the advent of the dormant season the fungus apparently ceases activities. It is not definitely known how the fungus passes the winter, but the supposition is that it hibernates as mycelium in lesions on the canes and mummied

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grapes. In the spring spores from acervuli in the old lesions bring about the first infections.

Moisture and the proper temperature are essential to the development of the pathogene. It is said that a poorly-drained soil also favors the fungus.

Control.

Until recently little has been done in American vineyards to prevent anthracnose, although its control is well understood in Europe. Recommendations based on the results of careful experimentation follow: (1) prune out and burn all diseased wood; (2) spray dormant vines with lime-sulfur, diluted one to nine; (3) spray the vines with bordeaux mixture, as for black-rot of grapes as follows: (*a*) when the shoots are eight to twelve inches in length; (*b*) just before the flower-buds open; (*c*) just after the blossoms fall; (*d*) ten to fourteen days later; (*e*) and again in ten to fourteen days. The addition of two pounds of resin-fish-oil soap to fifty gallons of the spray mixture in the last two applications is advisable on account of the increased adhesiveness of the fungicide which results.

For the dormant spraying lime-sulfur may be replaced by a fungicide of the following formula:

Iron-sulfate, 13¾ lb., or 6⅞ lb.
Sulfuric-acid (commercial) 7 oz., or 3½ oz.
Hot water, 3¼ gal.

This mixture of iron-sulfate and sulfuric-acid has been successfully applied in Europe. It is objectionable, however, for it is unpleasant to prepare and to use because of the corrosive action of the sulfuric-acid. A four per cent solution of sulfuric-acid has also been used as a substitute for other dormant fungicides.

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CROWN-GALL

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

This is the same disease which occurs on the peach, apple and other plants, a list of which appears on page 108. It is called crown-gall, crown-knot, root-tumor and black-knot (Fig. 66). Certain varieties are found to be more susceptible to the disease than others. In many localities the growing of varieties of grapes susceptible to crown-gall has become unprofitable. Susceptibility is believed by certain authorities to vary with the susceptibility to frost-injury; others hold that sap-acidity is a factor in determining susceptibility. Among the kinds resistant to crown-gall may be noted: Concord, Catawba, Delaware and other American varieties. European grapes are more susceptible, and some of these, arranged in

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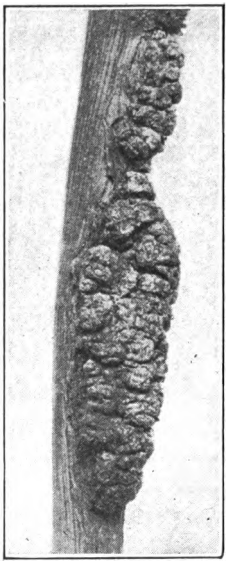

FIG. 66. — Crown-gall disease on grape-cane.

order of their susceptibility, follow: Muscat of Alexandria, Mission from California, Malaga varieties and Flame Tokay.

Crown-gall apparently affects the grape wherever it is grown. The disease is reported from France, Germany, Italy, England, Holland, Denmark, Chili, New South Wales, Cape Colony, Canada and Mexico. In the United States it has been found more particularly in California, New Mexico, Texas, Arizona, Washington, Oregon, Colorado, Arkansas, Nebraska, Missouri and Iowa. It was common in California as early as 1880. The earlier records give the impression that it was very destructive, but now the disease is not regarded as being of great economic importance. In cases of severe attack, vines are stunted, the leaves are smaller and chlorotic, and even unfruitfulness has been observed. (See more complete account under Apple, page 108.)

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DEAD-ARM

Caused by *Cryptosporella Viticola* (Reddick) Shear

Dead-arm, or side-arm, is found on nearly all commercial varieties of grapes in the eastern states, although it is rare on

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the Delaware variety. The Pocklington, although not extensively grown, is apparently most susceptible.

The disease appears to be of American origin, and there is little doubt but that it is still confined to this country. It was unknown until 1909, when its prevalence and cause were discovered. Losses from dead-arm are estimated at 1 to 5 per cent annually for New York vineyards, while the total for all states concerned is unquestionably of considerable importance.

Symptoms.

The arms, trunks, green shoots, petioles, peduncles, leaf-veins and fruits are affected. As the name indicates, the disease exhibits itself chiefly through the death of the arms (Fig. 67). Frequently, however, the whole vine dies, in which case suckers usually grow up at the base. The affected vines may die at any

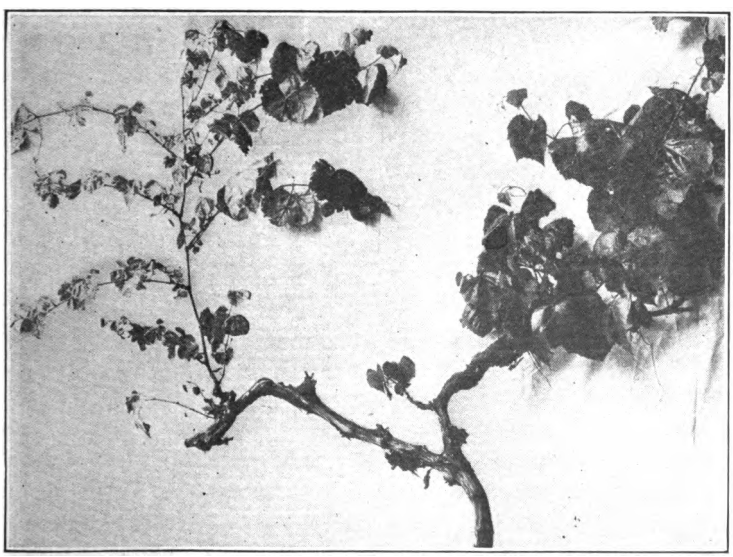

FIG. 67. — Dead-arm of the grape-vine, general appearance.

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time. However, the majority die in winter. Occasionally longitudinally ribbed excrescences develop on the trunk or arm of diseased vines. These outgrowths are not fleshy, nor are they hard, and they should not be confused with crown-gall (see page 253) and outgrowths following winter-injury. Internal symptoms are characterized by a dry heart-rot of the trunks in affected portions.

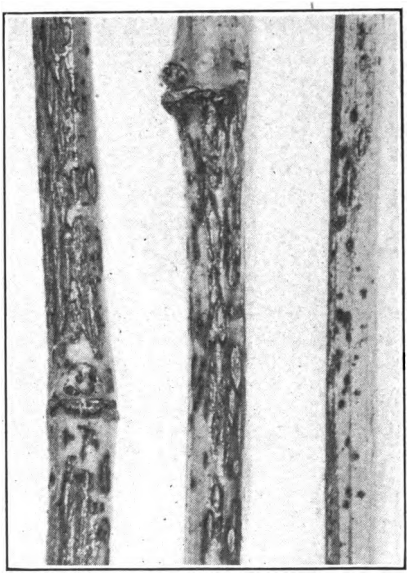

FIG. 68. — Dead-arm on grape-canes.

In June or early July the leaves show a peculiar yellowing, dwarfing and curling. Later, the discoloration disappears, although the other abnormalities persist. Such vines, while apparently on the road to recovery, are found to be dead or are considerably weakened the following year. The vines may die during the summer and as a result the leaves wilt. Many vines freeze to death as a result of a weak growth the previous season.

On the green shoots, petioles, peduncles and leaf-veins small reddish brown or black spots are produced (Fig. 68). These may be deep, in which case V-shaped slits are noticeable; or they are more superficial, and are so numerous as to coat the affected part for some distance. When these lesions are older, they show either as reddish elevations or as a longitudinal cracking.

Berries affected with the dead-arm disease exhibit a rotting

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(Fig. 69) very similar to that in the case of the black-rot disease. They shrivel to a mummy as in black-rot, but have a slightly more grayish aspect and the pustules are less numerous and more scattered in dead-arm than in black-rot.

Cause of dead-arm.

It has been experimentally demonstrated that the fungus *Cryptosporella Viticola* causes dead-arm. From vines which were diseased the previous year pycnospores ooze forth, during wet weather, about the time the buds burst in the spring. These spores are spattered promiscuously, some of them falling on young shoots, either those directly beneath the old infected cane or those some few feet away. After about thirty days symptoms of dead-arm begin to appear. During this period the spores of the fungus have germinated, the germtube has penetrated the part concerned, and infection has resulted. Days of mist and fog favor spore-germination, the process requiring not more than twenty-four hours. The manner in which the fungus gets into the vine is unknown.

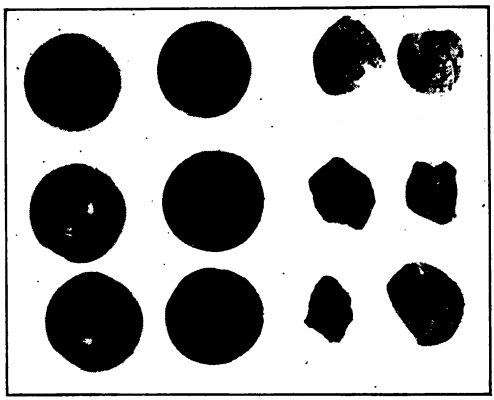

FIG. 69. — Rot of grapes caused by the dead-arm pathogene.

In a great many cases the mycelium developed from the germtube may grow from affected canes into the permanent wood of the spur, arm or trunk, although in cases of less severe infection this does not take place. The growth of the mycelium in the canes is very slow, but it gradually gets into the arms and

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trunks. When affected canes are saved for bearing wood, the fungus spreads into the arm. As a rule the mycelium does not extend into the roots. Pycnidia are developed abundantly on affected canes and on green and ripening berries (Fig. 70). They appear on the former early in the spring, and may be found on canes severely infected the previous year or occasionally on the current year's growth. The pycnidia mature shortly after the buds burst in the spring, and the pycnospores ooze out in long, reddish yellow coils, each containing several thousand spores. Rain favors this exudation. Perithecia are developed on a stroma beneath the bark, but these bodies do not seem to be important in the life-history of the fungus. They are rarely found, having been reported from but three southern states.

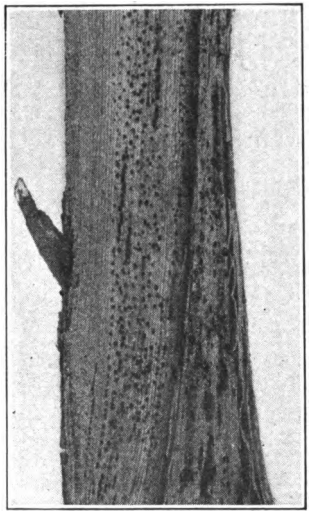

FIG. 70. — Dead-arm; fruiting bodies of the pathogene.

It has been shown that the transmission of the fungus through nursery-stock is of no little consequence. Badly affected cuttings do not root. Cuttings which show but few lesions will probably make vines which might grow for several years without exhibiting marked evidence of the disease. It has been shown further that infections may result from the pruning tools; in cutting through an affected vine bits of the fungus, which cling to the tools, are deposited in the cut next made on another vine.

Control.

In attempting to control dead-arm the following points should be remembered:

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(1) Vines which exhibit a dwarfed, yellowed foliage during the early part of the season (in June) may be regarded with suspicion; it is probably a case of dead-arm. Diseased plants are thus located.

(2) About this time the ground is being cultivated frequently; vines may then be marked and at trimming time may be removed. In two or three seasons all affected plants may be eliminated.

(3) The disease affects the heart-wood. If vines show this, they should be removed and the cut should be made at some point below the last indications of dry-rot. Often this is at the ground, in which case renewals come from below the surface.

(4) Renewals must be carefully inspected, for they may become infected from some outside source.

(5) At trimming, reddish elevated lesions should be watched for. More than passing attention must be given when dead-arm is present in the vineyard.

(6) Vines may be brought quickly into profitable bearing by the renewal system.

(7) It has been shown that the fungus may be carried by pruning tools.

Healthy vines may be protected from new infections by the application of bordeaux mixture. This should be done at the time recommended for the first black-rot spraying, that is, when the shoots are eight to ten inches long. The application should be made every year, regardless of the weather. For while practically no infections occur in certain years, yet such conditions cannot be safely forecasted by the grower. Nurserymen should take cuttings from vineyards which receive this early application or which are known to be free from dead-arm.

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White-Rot

Caused by *Coniothyrium diplodiella* (Speg.) Sacc.

It is probable that the pathogene causing white-rot is native to America, although it was first found in Italy about 1878. In 1885 it was observed in France, and in 1887 considerable alarm was aroused because of white-rot. That same year the disease was first noted in America, having been found in southwestern Missouri and neighboring territory. Since its discovery in the United States the disease has appeared in Ohio and New York and the grape-growing regions of the Southwest. In recent years white-rot has been found in Algeria and Hungary.

White-rot is regarded as one of the less important of the grape diseases in America. It is not often serious, although it is said to do damage in some parts of Ohio. The amount of losses from white-rot is decidedly greater in European vineyards than in those of this country.

Symptoms.

White-rot affects the fruit, young shoots, stems of berries, and rarely the foliage.

Berries may or may not be directly affected. Generally the disease appears first on the fruit-stalks, as a result of which the berries wither and become dry. Berries affected directly show brown-rot lesions. The fruit becomes abnormally juicy, shrivels, and brownish pustules appear over the surface. Berries may be affected after maturity. In severe cases all fruits in a single bunch may be diseased. Eventually a dry mummy is produced,

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but it neither blackens nor shrivels into prominent ridges as in the case of black-rot.

Cankers are formed on the peduncles. These are brownish, depressed areas which may enlarge continuously until the part is girdled. This results in a withering of the berry, as already noted.

Cause.

The causal fungus is referred to as *Coniothyrium diplodiella.* It is claimed by certain French authorities that at least one other form (sexual) exists in that country, and therefore the proper name should be *Charrina diplodiella.*

The mycelium of the fungus is abundant in the lesions, particularly those on the berries. Sometimes the seeds are affected. In the peduncles the mycelium causes the death of the tissues as a result of which the berries wither. About the time grapes begin to ripen pycnidia are produced. These arise as a result of a special growth of the mycelium; a cushion is developed in which a cavity bearing spores is finally formed. These fruiting bodies lie beneath the cuticle until maturity, when they burst forth, first appearing as shining, rosy points, then white and ultimately brown in color. Although perithecia have been reported in France, they are unknown in America.

Control.

It is doubtful whether special treatment for white-rot is ever necessary. The disease should be controlled by the black-rot spray-schedule; at least there is nothing to indicate the contrary.

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Shelling

Cause somewhat obscure

Grape-growers in certain parts of America have known this trouble for at least fifty years. It has been reported especially from Connecticut, central and western New York, and Michigan. What appears to be the same disease has been observed in Australia (Queensland) and in many parts of France. In France there appear to be two forms of shelling: one in which there is a failure of the flowers to set fruit in some or all parts of the cluster, known to the French as coulure; the second form, in which the fruits fall or shell. The latter type is the more important in this country; the losses at times being serious. Fifty per cent of the fruit may drop when affected with shelling, or rattling.

Symptoms.

Affected grapes fall two or three weeks before maturity. Those diseased berries, particularly of the green varieties, exhibit a peculiar though indistinct mottling of the surface. The skin becomes abnormally thick, and the whole berry is harder than healthy berries of the same age. The interior of such a fruit shows a brown zone just beneath the skin. The taste of shelling berries is noticeably insipid as compared to the tart, astringent flavor of the healthy, unripe berries. Shelling grapes separate easily from the stem, leaving the latter as if cut with a knife; no such phenomenon occurs with unaffected berries. Generally those berries at the lower end or at the extremity of the shoulder are first to fall from the bunch. Shelling is not always accompanied by foliage discolorations, nor is a browning of the leaves a certain indication of the disease.

Cause.

The cause of grape-shelling is obscure, although a great many suggested causes have been eliminated by close students of the trouble. Among the excluded primary causal factors

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may be noted: certain fungi, insects, lack of phosphoric-acid and meteorological conditions. Those agencies which are thought to increase or favor shelling are: a weakening of the vines due to overbearing, heavy vegetative growth, excessive nitrogen supply emphasized by over-tillage, prolonged drought or excessive rains followed by drought, and a poorly developed root-system. One authority concludes that a lack of potash is in many cases the primary cause. There is considerable evidence, however, that this is not a factor. The agencies already listed as favoring the trouble may be regarded as exciting causes.

Control.

Since the main cause of shelling is apparently deficient nourishment, steps should be taken to correct such conditions. The soil should receive attention; potash should be applied where needed. Cultivation of poorly-nourished vines aggravates shelling; proper cultivation is essential. Care should be taken to properly handle vines that overbear; such plants need an adequate food-supply. In France it has been advised that shoots be pinched or cut off after the development of six to eight leaves where the grapes are borne; or twelve to fifteen on those shoots not carrying fruit. This diverts the elaborated food from the shoots to the berries. This procedure is said to give satisfactory results in France.

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Ripe-Rot

Caused by *Glomerella cingulata* (Stoneman) Sp. and von S.

This disease is sometimes called anthracnose, but it should not be confused with the anthracnose disease discussed on page 249. The name bitter-rot is also used to designate this trouble, although ripe-rot is preferable.

Ripe-rot was known several years prior to 1887, at which date grape diseases first received the serious attention of American plant pathologists. The history of the disease shows that the first observations on it in the United States were made in North Carolina, although the pathogene was described in England in 1854. At present the disease has a general range over the grape regions of the country, but only occasionally is there any wide devastation. White varieties, like the Martha, are sometimes affected in a destructive fashion. A characteristic of the disease which makes it annoying and dangerous is that even after a crop has escaped other grape diseases during the year, ripe or ripening berries may be attacked to a considerable extent.

Symptoms.

Berries, canes and fruit-pedicels may be affected by ripe-rot, but it is most conspicuous on the berries. Ripe grapes only are affected; or at least they are not affected until the ripening period is near at hand. The diseased flesh becomes reddish brown or rosy in color and the surface is sunken. Finally the lesion, by enlarging in concentric zones, involves the whole berry, and the result is a brown or purple mummy. The lesions are more striking on light-skinned varieties. These characters serve to distinguish ripe-rot from black-rot. It has been noticed that ripe-rot mummies fall to the ground at the slightest jar, while black-rot mummies cling tenaciously to their pedicels. Affected berries are not bitter, as the name bitter-rot would suggest. The name bitter-rot has been adopted in the several

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cases on account of the fact that the same disease occurs on apples, on which it is known as bitter-rot.

Cause.

Ripe-rot of grapes is caused by the well-known fungus *Glomerella cingulata.* The fungus occurs on a wide range of plants, including many fruits. Its mycelium permeates the affected tissues, and even enters the seeds of the grape-berry. A cushion of mycelium develops just below the upper wall in a group of epidermal cells. As it increases in size the cushion —the fruiting body—pushes upwards, rupturing the epidermal walls. In this way the fungus exposes its fruit-body to the air; spores are developed and may be liberated with little hindrance. The pathogene may readily pass from the grape to the apple and back again.

The fungus is favored by excessive humidity and hence is most to be feared when such conditions prevail at the ripening period.

Control.

The lateness of the attack of the fungus on grapes makes it difficult to control. It is claimed, however, that if the later applications for black-rot are made thoroughly, the ripe-rot disease will be largely prevented. (See page 235 for black-rot control.)

More complete data concerning the fungus as it occurs on apple are given on pages 14 to 21.

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Brunissure

Cause not definitely determined

Brunissure is a name taken from the French which means a browning. The term refers to the effects on the foliage. The first manifestation of the disease is the appearance of irregular brownish spots on the upper surface. The epidermal cells finally slough off, exposing the underlying parenchyma, and numerous white spots appear on the dark background. Ultimately the browning involves the tissues through the leaf and the lower surface is reached. The trouble shows from July to November. As a result of the affection the berries ripen poorly, their sugar content is reduced, the vines fail to mature, and black zones appear in the wood as far down as the roots.

The disease is known in France, where it appeared in the autumn of 1892. It is also known in Italy and elsewhere abroad. Typical cases have been observed in the United States, notably in California. It is destructive only in isolated cases. The disease is thought to affect other plants such as the lily, tobacco, tomato and rose.

The cause of brunissure has not been positively determined. At first it was thought to be due to scale insects, then to a slime mold, *Plasmodiophora Vitis* (*Pseudocommis Vitis*). Unfavorable soil and weather conditions have been suggested as possible causes. It is generally agreed at present that the disease is non-parasitic, but this helps but little in disclosing the true cause. Control measures are unknown.

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California Vine-Disease

Cause unknown

Although this disease occurs in Italy, chief damage has been wrought in California. At one time it was the most serious of the several vine-diseases in that state. It first appeared there in 1884, as nearly as can be determined, and within two years many vineyards were destroyed in the vicinity of Anaheim, California. The destructiveness of the disease in this locality has given rise to the name Anaheim disease. Up to the year 1895 about 30,000 acres of productive vineyards were killed. The loss has been estimated at not less than $20,000,000.

Symptoms.

Except for the older pathologists who have known the disease from the first, few are able to recognize with accuracy the California vine-disease. When it first appears in a vineyard it shows here and there in a sporadic fashion. But in time it increases; it is cumulative. The leaves, canes and roots are affected. During the first season symptoms of the disease in a vineyard show on the foliage. Small yellow spots appear in the tissue between the veins. These enlarge and unite, forming yellowish stripes which broaden and die at the center. Eventually there is a conspicuous brown stripe of dead tissue bordered by yellow on each side, leaving only a narrow band of green tissue along the veins. Leaves fall prematurely, dropping from the base of the canes first. The immature portions of the canes turn black and die, and the berries become dry and hang to the vine, or rarely fall. The next season there is a reduced growth of the canes. Often the foliage will appear normal in the spring, but it becomes spotted during the summer, followed by a premature defoliation and death of the canes as described for the first season. The third spring the vine may fail to put out new growth, or it may grow until midsummer and then die. In some cases affected vines live for

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five years. The roots on diseased plants decay first at the tip, then other parts succumb, and finally the whole system is involved.

Cause.

The disease is not only destructive but obscure. As yet the cause is not known. It has been concluded by many authorities that it is of non-parasitic origin. They claim that overbearing is not an impossible cause. It is also held that the disease is due to some weakness in the functions of absorption and translocation of water. There is some evidence that the causal factor, whatever its nature, is transmitted by cuttings.

Control.

The use of cuttings from healthy vines from a district where the disease does not occur is advised. Healthy cuttings, however, may contract the disease if grown among affected vines.

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CHAPTER X

PEACH DISEASES

THE peach, although fairly well acclimated in the United States, is by no means exempt from fungous, bacterial and other troubles. It is a matter of common knowledge that the peach tree is short-lived. The relatively premature disappearance of these trees is brought to pass by various factors, among which diseases and injuries assume a prominent position.

Many of the best-known diseases, such as brown-rot and leaf-curl, are relatively easy to control; otherwise the life of a peach tree would be even shorter. But in too many cases carelessness has allowed these two diseases to go unimpeded. It is admitted, however, that in the warmer peach-growing states the brown-rot fungus has a decided advantage and the grower not infrequently is almost helpless, and certainly is handicapped. Leaf-curl is most troublesome farther north, particularly in localities where the springs are moist and cool. But experience has shown that in most years the proper application of recommended control-measures will reduce this disease to a profitable minimum. The control of brown-rot and leaf-curl is discussed on pages 275 and 282, respectively. The extreme sensitiveness of peach-foliage to spray-injury makes it imperative that care be taken in the selection and application of sprays.

The less important peach diseases are represented by scab, die-back, black-spot and Coryneum-blight. These are fairly well understood and are amenable to control in most cases.

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Peach-yellows, and similar diseases, like rosette and little-peach, are best known because of their destructiveness and obscure causal nature. The best authorities gave up the unsolved problem of their causes several years ago. And to-day these troubles remain in obscurity, at least in this respect. Control measures for these diseases are therefore puzzling and ineffective. In many localities peach-yellows is the most dreaded of all peach enemies. But for the whole United States brown-rot and leaf-curl are the most important diseases.

Brown-Rot

Caused by *Sclerotinia cinerea* (Bon.) Schröt.

This disease, which is called brown-rot of stone-fruits, mold, blossom-blight, twig-blight, peach-rot, brown-rot canker and other names, was not given serious consideration in America prior to 1881. It is now a well-known fungous trouble wherever the peach is grown, both in Europe and in the United States. The pathogene causing brown-rot probably came from some foreign country. History shows that the disease has been more serious in some years than others in America. In 1887 it attracted no little attention in Maryland and Delaware. In 1891, 1893, and during subsequent years brown-rot has been of considerable importance in the Delaware and Chesapeake peninsula. Alabama growers experienced a severe epiphytotic in 1897, while in 1900 the disease was the most conspicuous and the most destructive in Georgia since the beginning of stone-fruit culture in that state.

Brown-rot is most prevalent and most destructive in the warmer peach-growing states, such as have already been enumerated. In warm, wet seasons the trouble is severe in the northern states. The light-colored varieties are generally regarded as the most susceptible. Those showing least rot

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in Georgia are the Carman, Early Crawford and others. Observations on the question of varietal resistance need to be extended.

The average annual loss to the peach-growers of this country because of brown-rot is placed at $5,000,000. For the year 1900 Georgia growers are said to have lost from $500,000 to $700,000, and the average annual loss is estimated to be not less than 40 per cent. Again, in 1909 the loss of peaches due to brown-rot in Georgia, with only one-third of an average crop, is said to have reached $1,000,000. Almost a total loss of the peach-crop is reported from Alabama in 1897, and similar losses occur in other southern states. While in the northern states and in Canada peach-growers do not experience any such calamities, yet these regions are by no means exempt from the disease, and in some years the outbreak is severe. The loss from this disease is not limited to the fruit-growers, but is felt by the transportation companies, the commission men and the consumers. A shipment of fruit which shows great promise as it leaves the orchard may reach the market in a worthless condition. The disease is destructive in one or more of the following ways: (1) blossoms may be blighted, destroying

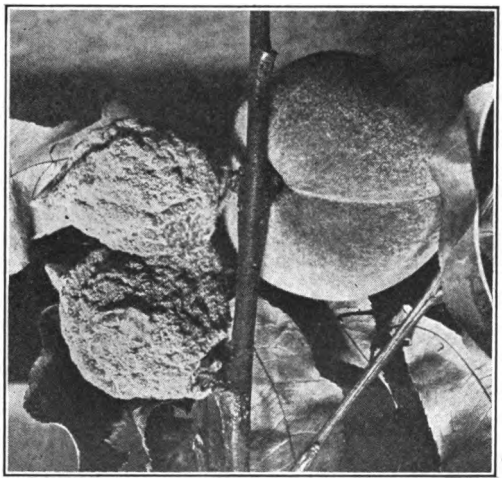

FIG. 71. — Brown-rot on peach.

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the set of fruit; (2) twigs may be blighted, thus inflicting serious injury on the tree; (3) large limbs may be cankered, which form of the trouble is of great importance in parts of New York State; (4) the crop of fruit itself may be partially or wholly destroyed as a result of attacks by the brown-rot pathogene; green fruits, if injured by insects or hail, may be rotted extensively; and likewise ripe fruits on the tree, in transit, or in market may be wholly ruined as a result of rotting.

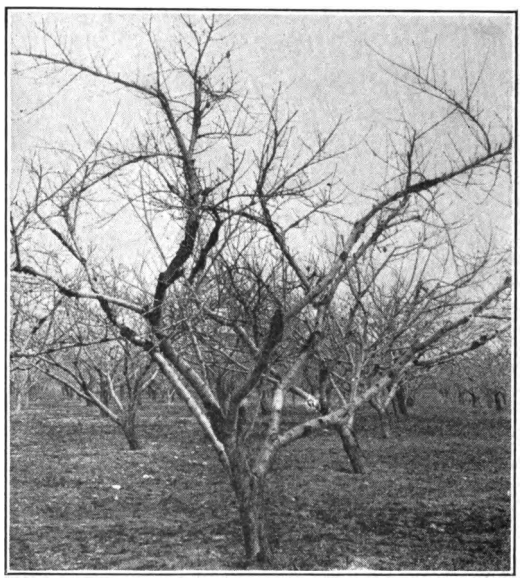

FIG. 72. — Brown-rot canker of peach, general appearance in the orchard.

Symptoms.

The fruits are most commonly affected (Fig. 71). However, other organs of the host are also susceptible to the disease; these include the blossoms, twigs, limbs (Figs. 72 and 73) and occasionally the leaves.

In America the flowers are commonly affected, resulting in a serious blossom-blight. When the blossoms are opened, the petals turn brown and shrivel, but do not fall. Grayish tufts composed of the fruiting structures of the pathogene show on the affected blossoms. Twig-blight follows blossom-blight very closely, the former being a direct result of the latter. The leaves on such twigs wither and die and cling to the twig,

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as in the case of fire-blight of pears. Twig-blight may also result from the spread of the pathogene from affected fruit into the twig by way of the fruit-pedicel. This happens very commonly in New York State orchards. From the twig the causal pathogene passes into the larger limb where it spreads out, forming a canker (Figs. 72 and 73). The brown-rot canker is a definite dead area in the bark, the surface is sunken, and the lesion is accompanied by a flow of gum (Fig. 73). The disease is said to affect peach leaves, showing itself as a shot-hole.

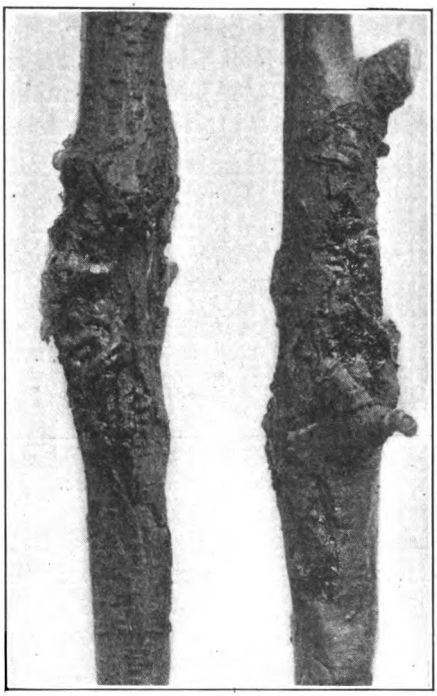

FIG. 73. — Brown-rot canker on peach limbs.

Fruits generally show signs of brown-rot after they are half grown, the susceptibility of individuals increasing as they approach maturity. The lesion on the peach is at first evident as a small, more or less circular, dark-brown, decayed area with a rather indefinite line of demarcation between the healthy and diseased portions. This rapidly enlarges, and soon the fruiting structures of the pathogene appear on the surface as grayish tufts (Fig. 71). At first these tufts occur sparingly, but in a day or so the original rotted spot may become densely dotted with the characteristic ashen mold (Fig. 71). With

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the enlargement of the lesion the whole fruit becomes involved, is brownish, shrunken, and eventually shrivels into a dark mummy. The mummy may cling to the tree or it may fall to the ground. While on the tree mummies cling together in groups of two or more.

Cause of brown-rot.

The pathogene, the fungus *Sclerotinia cinerea*, hibernates in both the fallen and hanging mummies, and in the cankers. From sclerotial crusts in the fallen mummies arise apothecia (Fig. 74) in the spring of the year. The hanging mummies furnish a habitation for conidia through the winter; in the spring these spores are carried to the susceptible parts. These old clinging mummies as well as the cankers also contain mycelium of the fungus, and in the spring conidia are developed from these sources. The source of the primary inoculum is found, then, in both the fallen and hanging mummies, and in the cankers. The inoculum itself consists of both ascospores and conidia, of which the latter are the more important. In fact, it sometimes appears that ascospores are not developed every year, and that they play a very minor rôle in the life-history of the brown-rot fungus. Spores of either kind are carried to the blossoms, where blossom-blight is induced. The mycelium grows throughout the blossoms, enters the twigs, and finally passes into the large limbs. Conidial tufts develop on the petals, and even on the cankers and blighted twigs, if the atmosphere has a high relative

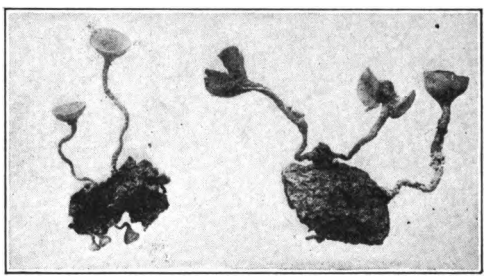

FIG. 74. — Apothecia of the brown-rot fungus; attached to fallen mummies.

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humidity. The conidia from these sources start the disease in other parts of the peach. From one or more of the several possible sources conidia are carried to the half-grown fruits. These spores germinate, and the tubes enter through an injury; the curculio and the peach-scab fungus are the chief agents in making such wounds. The mycelium develops profusely within the tissues of the fruit, the result showing externally as a brown-rot. Conidial tufts soon appear and conidia are liberated to continue the destructive action of the fungus. Where two or more peaches touch each other, the mycelium grows from the infected peach through the point of contact to the other. In this way the fruits are made to cling to each other, several in a group.

The fungus is highly favored by a series of cloudy days accompanied by frequent showers, especially at picking time; consequently a great amount of fruit may be destroyed under such weather conditions. Prolonged drizzly weather is far more dangerous than a heavy rain followed by clearing. Hot weather favors the rapid growth of the fungus and increases the danger of its destroying the crop. On the other hand, in a dry, cool season the crop may be expected to remain relatively free from brown-rot.

Control.

In applying control measures it should be borne in mind that: (1) all parts of the peach above ground are liable to attack; (2) the disease is most serious in warmer regions and in warm, wet seasons; (3) total destruction of the crop may come suddenly and unexpectedly; (4) the fruits and blossoms are affected seriously, which fact is of vital importance when measured in dollars; (5) the fungus overwinters in the mummies and in the cankers, and the mummies are found both on the ground and hanging to the tree; (6) the fungus enters fruits chiefly through wounds, and the curculio is a serious offender in making such wounds. Therefore, remove all

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cankers from the larger limbs, and prune out affected limbs of smaller size. The methods followed are described under another heading (see page 52). In pruning, knock off the hanging mummies. Bury the fallen mummies by plowing to a depth of several inches; this should be done before the blossoms open. The regular pruning operations assist to no little degree in the control of the brown-rot. The removal of limbs admits sunlight and air, thus allowing the susceptible parts to dry off more quickly after rains.

The fruit should be sprayed. This can be done without fear of injury to the foliage by the use of self-boiled lime-sulfur of the 8–8–50 formula (see page 438). For curculio, add 2 pounds of lead arsenate to 50 gallons of the fungicide. Never use lime-sulfur solution as a summer spray, even at dilute strengths, since a 1 to 300 dilution may seriously burn peach-foliage. Spray: (1) about the time the blossoms fall; (2) two or three weeks later or about one month after the petals drop; (3) about one month before the fruit ripens.

Limited experiments indicate that lime-sulfur solution 1–40 may be used safely and effectively before the blossom-buds open. This application purposes to prevent bud-infection, and is worthy of trial.

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Leaf-Curl

Caused by *Exoascus deformans* (Berk.) Fckl.

It is evident that this disease has long been associated with the peach, this fruit-tree being a natural host for the causal organism. Furthermore the disease is distinctly one affecting the peach and its derivatives, such as the nectarine and peach-almond. It has rarely been found on other fruits. This trouble, known as peach leaf-curl, curl, curly-leaf and leaf-blister, affects both orchard trees and nursery stock, certain varieties showing more resistance than others. The Elberta and Carman are the most susceptible in New York State, while, in the nursery at least, the Richards is resistant. Seedling peaches show marked susceptibility, which fact indicates that the disease may have originated with the wild peach in central China.

The disease has long been known in England, where it was described as early as 1821 by an English gardener as blister. It has become widely distributed throughout the world wherever peaches are grown, being most serious near the seacoast or in the region of large interior lakes. In the United States and Canada it is very common and destructive in the peach-growing sections about the Great Lakes. Likewise in the Pacific

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Northwest it has been very troublesome in regions of considerable annual rainfall.

Peach leaf-curl is regarded as the most serious fungous disease affecting this fruit in cooler climates. In warmer climates, on the other hand, brown-rot assumes this rôle. The fruit being rarely attacked by the leaf-curl pathogene, the losses involved are usually underestimated. The annual toll which American peach-growers give to the ravages of this pest is said to be three millions of dollars. The character of the losses is: (1) loss of leaves in the spring, followed by a forcing of a new crop of foliage later that year, which lowers the vitality of the tree; (2) partial or total failure of trees to set or hold a crop because of defoliation; (3) repeated loss of leaves for several seasons in succession, resulting in the death of the trees; (4) injury to trees by killing the twigs; (5) stunting of nursery stock due to the death of the shoot from the bud; curl-affected nursery buds never make good trees.

Symptoms.

Most peach-growers are familiar with the symptoms of peach leaf-curl, particularly in its later stages (Fig. 75). The first evidence of the disease, however, may be frequently overlooked. In the spring shortly after the leaves begin to unfold there is a puffing and folding of these organs. The leaf-blade becomes thickened and puckered along the midrib, causing the leaf to curl. The diseased portion becomes yellowish, with tints of red. The leaf is thickened, becomes brittle, and finally shows a characteristic silvery bloom over the upper surface (Fig. 75). Curling may be confined to a part of the blade, or the petiole, or may involve the whole leaf (Fig. 75). Affected leaves finally die and drop from the tree, in some cases the entire tree being defoliated. A new set of leaves is then formed from the dormant buds following defoliation. Affected twigs show a marked swelling and are stunted in length. Their color changes to pale-green and yellow. While the hypertrophy

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FIG. 75. — Leaf-curl of peach.

involves the current year's growth for the most part, sometimes the pathogene extends down the side of the previous season's growth, forming a swollen ridge. Badly infected twigs are

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usually killed, but the growing tip may develop a healthy shoot, leaving a swollen canker-like lesion at its base. An exudation of gum often accompanies the lesions on the twigs. The flowers and young fruits are often attacked, but because they soon drop away this symptom of curl is seldom observed. In general, the sickly yellow, curled foliage, and the final defoliation of the trees the latter part of June, followed by a refoliation, are the most striking symptoms of this disease.

Cause.

The peach leaf-curl disease is caused by the fungus *Exoascus deformans*, so named because it forms its asci on the outside of the host, and because it deforms the leaves. The mycelium of the pathogene grows between the cells of the leaf, stimulating them to abnormal increase in size and number, and robbing the leaf of its green chlorophyl. When the spores are to be produced, the mycelium invades the cuticle of the leaf, and at once gives rise to sacs (asci) which bear from 3 to 8 ascospores. The presence of the asci on the upper surface of the leaf gives the leaf its silvery appearance. At maturity, the ascospores bud extensively within the ascus, giving rise to spores suggesting conidia. The ascus then empties its contents through a crack at the top. Thus far the life-history of *Exoascus deformans* is definitely known; but where these ascospores and their budded descendants go is unknown. The fate and habits of the fungus during the summer, fall and winter are not clearly understood. It has been held that the mycelium is perennial in the twigs, growing out into the leaves and young shoots in the early spring. But this is doubtful, since thorough spraying in the fall or before the buds open in the spring will control the disease. Since the fungicide cannot penetrate the twigs to kill the fungus, it can only be surmised that the inoculum comes from some external source. It is the opinion of most investigators that the fungus hibernates on the bud-scales in the form of spores (kind unknown), and that these spores germinate during

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the spring rains at a time when the buds swell, and that the germtube penetrates the very young leaf as it emerges from the bud. This opinion is based on the following circumstantial evidence: (1) lesions appear on leaves just as they are protruding from the buds; (2) leaves from buds sprayed before such buds swell show little or no infection during the season, while unsprayed buds on the same tree curl badly; (3) buds sprayed after they swell, and especially after rains, show curled leaves; (4) the disease occurs only during cold, wet springs.

From the above data it may be said that, theoretically, some sort of spores are lodged by the wind or rains among the hairs of the bud-scales during the late summer, and that these spores remain there dormant until conditions favorable to infection arise the following spring.

The effect of the environment upon this disease is very marked. This is noticed in a general way in connection with the geographical range of the disease, which is most common and severe in the neighborhood of large bodies of water. The combination of conditions most favorable to an epiphytotic of peach leaf-curl is a cold wet-period following warm spring weather. Warm weather starts the buds; a cold wet spell immediately following results in a saturation of the leaf-tissue with water, due to a lowering of the temperature and to the high humidity of the atmosphere. The buds are retarded, their cells gorged with water, and their walls distended, while the damp atmosphere permits spore-germination and infection. As already noted, curl is most severe near large bodies of water; this is doubtless due to the increased humidity of the air in these localities, and to reduced temperature in the early spring as evidenced by the more frequent occurrence of fogs in such regions. Heavy dews can exert but little influence on curl, since the moisture and temperature factors are not sufficiently pronounced nor of adequate duration to effect a response on the part of the host and the parasite. Rainfall seems to have

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considerable influence on the disease. In the Pacific Northwest, for example, little or no curl exists east of the Cascade Mountains, where the annual rainfall is light; on the other hand, west of these mountains the rainfall is heavy and curl is very destructive. In these two regions the temperature is approximately the same.

Control.

The time and thoroughness of the application of remedial measures are important points in the control of peach leaf-curl. It is imperative that such work be done before the buds swell in the spring. Conjectures with reference to the winter and spring activities of the pathogene lead one to infer that spraying should prevent the disease. It has been shown that the application of almost any common fungicide ordinarily controls the trouble satisfactorily. One spraying is sufficient and may be made in the fall, or in the spring before the buds swell. Never spray more than once, as it is a waste of time and materials: and do not spray for curl after the leaves are expanded, for such an operation is useless and dangerous. Spray thoroughly, making sure to coat every bud. If the season is favorable to curl, the unsprayed buds will show the disease. Ordinarily lime-sulfur solution of standard Baumé test, 32°, at a strength of 1 to 15 or 1 to 20, is effective. If San José scale must be combated, both troubles may be controlled by using the fungicide at scale strength, 1 to 8. Scale can be controlled as effectively by spraying in the fall as in the spring. Bordeaux mixture of any strength, or copper sulfate 2 pounds to 50 gallons of water, are also effective in controlling peach leaf-curl, and may be used when scale is not a factor to be considered.

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YELLOWS

Cause not known

Wherever this disease occurs it is known as yellows, or peach-yellows. It is primarily a trouble of the peach and nectarine, although it has been observed on almonds, apricots and Japanese plums. Seedling peaches are said to be more sensitive than budded trees. Seedling trees derived from budded fruit are equally susceptible.

The origin of the trouble is unknown. Records of it date back to 1760, and in 1806 it was the subject of horticultural writings. The first discovery of peach-yellows is thought to have been made in Pennsylvania, near Philadelphia. Outbreaks of the disease occurred in the East in 1791, 1806, 1807, 1817 to 1821, 1845 to 1858, 1874 to 1878, and in 1886 to 1888. It appeared in Michigan about 1869, or earlier. The disease is not known on any other continent than North America, and the early records coming from the eastern part of the United States would, therefore, seem to show that peach-yellows is native to eastern United States. It occurs as far south as southern Virginia, west to Arkansas and northeastern Texas, north to Canada, and east to New England.

Peach-yellows is conceded to be one of the most serious diseases of the peach in America. It has been extremely ruinous in the region outlined in the preceding paragraph. Soon after its discovery, hundreds of orchards along the Atlantic Coast were destroyed by the disease, causing growers in many localities to abandon peach-culture. In one county in Michigan

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the number of peach trees destroyed by yellows between the years 1874 and 1890 is estimated at more than one-half million.

Symptoms.

The marked symptoms of peach-yellows consist (1) in a premature ripening of the fruit (Fig. 76); (2) a red spotting on the surface of the fruit; (3) the development of secondary shoots in great numbers (Fig. 77), these being dwarfed and unhealthy in appearance; (4) the development of short shoot-axes with sickly foliage of a yellowish or reddish brown color, having a tendency to roll sidewise. These symptoms begin to develop in the middle of the summer. Subsequent developmental stages are observed at various seasons of the year.

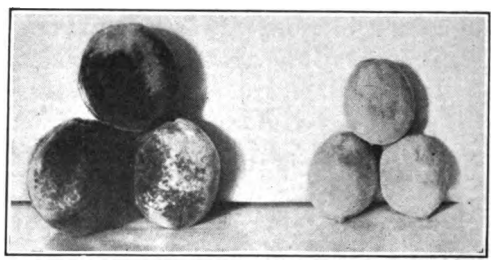

FIG. 76. — Peach-yellows; on the right small healthy peaches, on the left large prematurely ripened fruit.

Certain evidences of yellows are apparent the first year that the orchard is affected. (1) There is a premature ripening of the fruit (Fig. 76); this will likely be the first noticeable symptom. This premature ripening may take place a few days to several weeks prior to the time of normal ripening. It may occur on but one or two branches, in which case the peaches on the rest of the tree ripen normally, or all peaches on an affected tree may exhibit this abnormality. This phenomenon should never be confused with the work of the peach-tree borer; trees severely injured by this insect may ripen their fruit prematurely, but the flesh is never red spotted and shoots are not put forth as in yellows. (2) Diseased fruits are always reddish or pur-

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plish spotted externally, and they show red streaks scattered through the flesh. The amount of redness varies; there may be only traces, or the whole fruit may become entirely crimson. The flavor of such fruit is usually insipid and sometimes bitter. (3) Abnormal dwarfed shoots come out on the trunks and limbs (Fig. 77). These may not show, however, until the second year, in which case the only signs of yellows the first

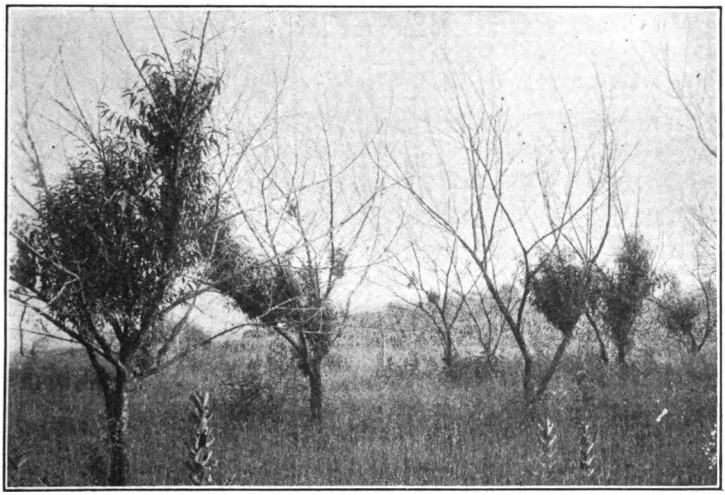

FIG. 77. — Peach-yellows; note the clusters of shoots.

year are those enumerated above. Such shoots bear small leaves of a pale-green, yellowish, reddish or whitish color, and there is a marked tendency toward repeated branching. Leaves on affected trees are notably more slender, but it should be borne in mind that certain varieties like the Elberta, Carman, Champion, Hill's Chili and others possess leaves which are characteristically long, narrow and straight, with a natural tendency to droop.

The second year during which the orchard is affected with

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yellows certain characteristic symptoms are exhibited. (1) Abnormal dwarfed shoots may appear on the trunks or limbs; but, as already noted, these may develop during the first year. (2) Affected trees may be barren after the first year, or they may bear another crop of fruit which ripens prematurely as described for the first year. (3) Affected trees may die the second year, but ordinarily they succumb slowly from the top downward (Fig. 77). When the first symptoms of yellows appear in any part of a tree, it is thought that the whole tree is then diseased.

The true yellows of peach should never be confused with little-leaf or California-yellows. This disease is characterized by the development of spindling, yellow shoots on the new growth with small, narrow, yellow leaves. The foliage along such shoots drops prematurely, leaving tufts at the ends. The fruit fails to develop; it shrivels and finally falls. It is a trouble peculiar to trees from three to seven years of age, whereas true yellows affects older orchard trees.

Cause of yellows.

Just what causes peach-yellows is still unknown. It therefore falls into a class, from the causal standpoint at least, with rosette and little-peach. The cause of yellows has aroused much speculation because of the importance of the disease and on account of its obscure nature. Many theories have been advanced to explain the origin of the peculiar symptoms exhibited by diseased trees, but all of these have been certainly disproved. Some of the prominent theories regarding the cause of peach-yellows follow: (1) severe winter-injury; (2) excessive rainfall; (3) impoverished soil, that is, a deficiency in lime, potash and phosphoric-acid; (4) insects; (5) fungi; (6) bacteria; (7) crowding trees in the orchard; (8) excessive cultivation; (9) over-bearing. Many others might be added. It will be seen that these fall into one or the other of the categories: weather, soil, orchard management and parasites.

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The prevailing opinion of the modern pathologists is that the disease is caused by a parasite which as yet has not been seen. It was long ago established that the disease is communicable, that is, the causal factor may be transferred from a diseased tree to a healthy one, and after a time the latter tree will show symptoms of yellows which in due time run their course. Buds from diseased trees convey the causal factor of yellows to the stock on which they are inserted. The inoculum, whatever its nature, is apparently carried in a few diseased cells which, if induced to unite with normal cells of the stock, will cause the stock to become diseased. Diseased seeds also carry the inoculum; and there are probably other ways in which it is carried.

Control.

The knowledge of remedial measures is about as meager as that of the causal relations. "Cures" are worthless and impossible. It has been shown that while fertilizers make the trees temporarily greener and apparently more vigorous, yet in the end they are of little or no value in the control of yellows. Trees have been treated with such materials as lime, wood ashes, kainit, muriate of potash, dissolved bone-black, bone-ash, nitrate of potash, nitrate of soda, sulfate of ammonia, tobacco dust, dried blood and stable-manure. Treated trees are just as likely to be attacked as those left untreated.

The prompt removal and destruction of affected trees has been recommended since 1828. The advice is still reliable. It has been practiced effectively in the State of Michigan in years past. The eradication of diseased trees must be an annual operation until no traces of yellows remain. Where no such efforts have been made to exterminate the disease, it has prevailed to such an extent that orchards have been destroyed and the culture of peaches abandoned. In order to accomplish general and effective eradication, laws have been enacted in many states. The first was enacted by the legis-

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lature of Michigan in 1875, which law made it a misdemeanor to neglect the destruction of diseased trees. This step toward legislation in general was followed by Ontario in 1881, and by New York in 1887. While laws exist in most states at the present time, they are ineffective because of a lack of enforcement; there is either a lack of personal responsibility or a lack of prosecution by authorities. Oftentimes many influential growers, being ignorant of the destructiveness of yellows, oppose the extermination of diseased trees. Frequently inspectors are not competent. In some states growers have no chance to appeal to the decision of inspectors.

Healthy trees may be reset on the same ground without danger. At least such trees are no more likely to be affected than their neighbors. Trees should always be purchased from a reliable nursery.

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LITTLE-PEACH

Cause not known

Like peach-yellows this disease is confined to the northeastern United States. It has been known for but a few years in this country. The origin of little-peach is unknown, but the suggestion has been made that it came from abroad with Japanese plums. The first appearance of the disease in America was once believed to have been in Michigan prior to 1893, but there are some indications now that it appeared in New York State at an earlier date. More recently important notices of little-peach have come from New Jersey and from Ontario, Canada.

This disease is not confined to trees of any particular age, although a larger number of the cases are found on trees over five years of age. In New York State the Smock and Salway varieties remained apparently resistant for several years, but finally these have become affected. In some localities of the states of New Jersey and Michigan little-peach is a more destructive disease than peach-yellows.

Symptoms.

Little-peach may or may not appear on a tree at the same time as yellows. In the former case the two diseases are often considered together. Little-peach is more difficult to detect than yellows. In the case of yellows the premature ripening of the fruit, the internal red splashes, and the slender shoots with narrow leaves are easy marks of distinction. But in little-peach, foliage characters are the most prominent earmarks of the disease, hence a knowledge of varietal characteristics of the foliage is necessary in detecting the presence of this trouble. For example, the Elberta has a long, wide, straight leaf with a

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drooping inclination, while the Crawford has a short, wide, crescent-shaped leaf which stands at right angles to the twig.

The first evidences of little-peach appear comparatively late in the growing season. It is still evident late in September. The fruit, instead of ripening prematurely as in the case of yellows, remains small and ripens about ten days later than is normal for the variety. The flavor of affected fruit is inferior, although the color may be as desirable as in healthy fruit. The flesh is characterized as stringy, especially in the case of early clingstone varieties. The pits of such fruits are smaller and shriveled, and almost invariably fail to sprout. Leaves on affected trees exhibit a light or yellowish green color, and those at the base and through the center of the tree show a rolled and drooped aspect. In severe cases all leaves on a tree show this symptom. This calls to mind certain stages of the yellows with which the little-peach disease may be confused, and unless the tree is bearing fruit at the time of the diagnosis it is difficult to determine which disease is present. However, the distinction between the two troubles is of no practical importance, and neither yellows nor little-peach is likely to be confused with other diseases. It is difficult to detect little-peach in young trees, but a possible character has been suggested, namely, a decided erect growth of numerous short twigs. Affected trees usually die within three or four years after infection.

Summarizing the prominent symptoms of little-peach, the following should be noted: the fruit remains small, about one-half or one-third its usual diameter, it ripens about ten days later than normal fruit, and possesses a bitter flavor and a stringy flesh; the leaves are small, light to yellowish green, and droop or incurve to some extent.

Cause.

This phase of little-peach has been the subject of no little investigation, but practically no progress has been made. The

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cause is not known, but it is supposed that the factor here involved is closely allied to that of peach-yellows (see page 286), but that the two diseases are distinct and entirely independent of each other. The disease is contagious; the causal factor can be carried by budding, although the disease may not come into evidence until the second year after inoculation in this fashion.

Control.

So far as known, diseased trees have never been cured. Spraying, watering, mulching and fertilizing have been of no avail. The removal of diseased parts is not effective, as it is in the case of fire blight of pears and similar diseases. The only remedy known is the removal and burning of diseased trees. To delay this operation only means added loss. This procedure, if pursued persistently, if all affected trees are systematically marked and destroyed at the proper time, will yield satisfactory results. The best orchard practice should be employed; favorable sites should be selected, only trees from reliable nurseries should be planted, reasonable cultivation, fertilization, spraying and pruning should be done. Observe the behavior of all trees and remove suspicious ones. Nurserymen should use care in bud-selection, taking every precaution against the disease.

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Rosette

Cause not established

The rosette disease affects many kinds of peaches, both budded fruit and seedlings. Probably the same disease occurs on many varieties of plums and almonds.

The disease was first described as a probable southern variation of peach-yellows, but later it was referred to as a distinct disease. The earliest record dates back to 1879, when it was observed in the State of Georgia. Ten years later it appeared in Kansas. It has subsequently been reported from South Carolina, Arkansas and elsewhere.

Affected orchards are lost within a few years. The trouble is regarded as second to peach-yellows in point of obscurity and destructiveness. The rosette disease is more rapid in its destruction than is yellows, and in Georgia, a prominent peach state, it has a wider range than yellows.

Symptoms.

Peach-rosette is recognized by the development of a rosette or whorl of sprouts on affected limbs. The whole tree, or only one or two limbs, may be diseased. Within six to twenty-four months the tree dies. First evidence is noted in the early spring when the buds open. Instead of only a few winter buds pushing out, a great majority of them grow into shoot-axes in compact tufts or rosettes. Such are only two to three inches long, and they bear numerous small leaves. When older and larger leaves are affected, they show an inrolling of the margins and a peculiar stiffening due to the midrib becoming straight. Smaller leaves are seldom rolled. Affected foliage turns yellow early in the summer and falls prematurely. Often leaves are blotched, browned and deadened at the ends and margins from the attacks of secondary leaf fungi. Fruit on rosette trees falls prematurely. Diseased trees show less tendency to develop sprouts on the trunk and main limbs.

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Peach-rosette differs from yellows in the more tufted character of the growths; in the absence of premature ripening of the fruit; more rapid destruction; and usually rosette is confined to the tips of branches, occurring but rarely on trunks and at the base of main limbs. It resembles yellows in the pushing out of dormant (adventitious) buds which develop diseased branches; the dormant buds tend to unfold in the summer and autumn; and only a portion of a tree may be affected, whereas the remainder is normal.

Cause.

This disease does not seem to be due to plant or animal parasites, nor to any chemical ferment. On the other hand, there is the slight possibility that it is caused by a bacterium which as yet has not been seen. The disease is contagious; the causal factor, whatever it may be, is readily spread by bud-inoculation, by root-grafting, or by blowing leaves. There are doubtless other ways by which it is spread, but as yet they have not come to light. Within two to ten months after a tree is inoculated first signs of the rosette disease are apparent.

Control.

The prevention of rosette is not known to be possible. All affected trees should be promptly destroyed. This should be done early in the spring as soon as the disease appears, and before the leaves fall. In those regions where the disease is present, attention should be given to the plum, which at times shows symptoms of rosette similar to those described for the peach.

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Scab

Caused by *Cladosporium carpophilum* Thüm.

This disease, known as peach-scab, freckles and black-spot, was first described in lower Austria in 1876. Since then it has been known commonly in the United States, and it occurs to an injurious extent wherever peaches are grown east of the Rocky Mountains. It is known also in California and occurs to some extent in Canada.

The damage done by peach-scab is apparently not realized by growers, some regarding the disease as a necessary evil, scarcely apprehending that their fruit is bringing in the market 25 per cent less price than clean fruit would command. Sometimes the disease is mistaken for a peculiarity of the affected variety. In spite of these misconceptions many growers do appreciate the importance of peach-scab and that it is the cause of widespread injury to the peach-crop. There is no decay of the fruit, but its market value is lowered; the size is reduced and the fruit dwarfed; the fruit is sometimes cracked, allowing rot-producing organisms to enter the flesh to cause subsequent rapid decay. Affected fruits may drop prematurely, and those which are picked do not ship well. In some seasons the loss, in Indiana for example, has been estimated at ten per cent of the crop, while in the eastern United States the loss has been put at the same figure. The total annual loss has been placed at $1,000,000 in the United States. The growing of certain susceptible commercial varieties has been prohibited by this disease. Heavy losses occur in West Virginia and western Maryland. In Ohio, in 1896, cases are recorded where 20–50 per cent of the crop was lost, while in New Jersey as much as 75 per cent of the fruit has been known to be affected in certain localities. In central and southern New Jersey the trouble is considered by peach-growers as one of their worst foes, while in the hilly portions of the northern part of the state, the disease

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does not cause injury sufficient to make spraying for its control a profitable or necessary operation.

Symptoms.

The fruits, leaves and twigs are affected. On the fruit (Fig. 78) small, round, olive-black spots begin to show about six weeks after the blossoms fall, or from June 15 until July 10, depending on the variety. Lesions most frequently occur on the upper side of the fruit. If the spots are numerous, they coalesce, forming a large, irregular diseased area covering a greater portion of the fruit's surface (Fig. 78) and preventing the normal expansion of the skin as well as interfering with

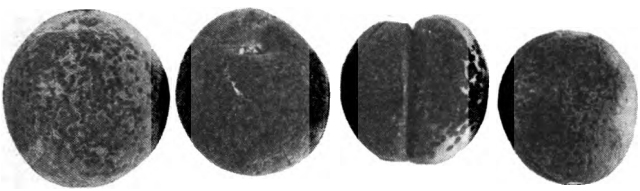

FIG. 78. — Peach-scab; types of lesions on the fruit.

the ripening and mellowing of the flesh. The fruit often becomes one-sided, due to a formation of a protective cork-layer under the diseased area. This layer is incapable of further growth, and hence an ill-formed fruit results. Often the cork-layer is ruptured, leaving deep cracks (Fig. 78) through which the brown-rot pathogene enters (see page 275). Attacks are generally most noticeable on the late varieties, due, in part perhaps, to the fact that the fruit of such varieties is exposed to infection over a longer period. Of the commercial varieties, the Heath is said to be most susceptible. The Bilyen and Hill's Chili are also badly affected, and the Salway, Smock and Morris' White suffer severely. The Elberta is sometimes badly diseased, but is more resistant than those just mentioned. The Carman, Hiley, Champion and Belle are slightly affected. Trees

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grown in higher places are more free from scab than those in low areas.

The twigs have more or less circular blotches of a yellowish brown color, with a dark-gray or bluish border. Frequently the spots become confluent, masking the normal pink-brown color of the young bark. The cuticle is separated from the cells beneath, corky cells are developed under the lesion, and in some cases the cambium is killed, and as a result the twig dies.

That portion of the leaves of the peach lying midway between the main veins is especially affected. Brown, scattering spots are developed in which the tissue dries up and finally falls away, leaving circular holes.

Cause.

Peach-scab is caused by the fungus *Cladosporium carpophilum*. Its mycelium occupies the space left by the partial separation of the cuticle from the underlying cells. On the twigs the mycelium hibernates in the form of dark-brown spherical cells. It is possible that conidia lying about the lesions may also be capable of tiding the fungus over winter. From the resting-cells described above, conidia are produced in the spring, and the latter are carried to the leaves and fruits. Germination follows shortly and penetration is effected. Inoculation begins four to five weeks after the petals fall, but symptoms do not show to the naked eye for about three weeks subsequently. Inoculations and infections continue to take place until about one month before the fruit matures. As the fungus grows on the fruit the mycelium attaches itself closely to the surface between the hairs, forming a mat of short, plump cells which give rise to conidiophores and conidia. The flesh of the peach is not penetrated, but the close contact of the fungus with the outer cells allows absorption of nutrition from the fruit through the unbroken walls. Evidently there is some injury to the outer cells, for a cork-layer is developed just

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beneath the lesion, suggesting an attempt on the part of the affected cells to repair the injury. Some time during the growing season the fungus infects the twigs, developing lesions as previously described. In these diseased areas the pathogene passes the winter.

Control of peach-scab.

In the early history of the disease, peach-scab was less amenable to treatment than it now is, because of severe injury of fungicides to the foliage. With the development of self-boiled lime-sulfur it has become possible to control scab without injury to the fruit or foliage. Where scab alone is to be treated, use self-boiled lime-sulfur 8–8–50 as follows: (1) four to five weeks after the petals drop; (2) about three to four weeks later. The second application is usually unnecessary if the first is thorough. Use about one-half to one gallon on trees three to four years old, depending upon the type of nozzle. The cost of one application on four-year-old trees has been estimated at four cents a tree. Spraying as directed above has been known to give from 92½ to 99 per cent of the crop free from scab.

Most peach-orchards in eastern United States should be given treatment for brown-rot as well as for scab. It should be remembered that preventing scab is an important step in the control of brown-rot.

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Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

Although regarded as harmful wherever it occurs, the best authorities have raised the question whether real injury results from crown-gall. Its progress is slow, and cases are on record where trees affected in the nursery, when planted in the orchard, made productive and profitable trees. On the contrary it is held that if a peach is affected at planting, it will never fruit successfully, and will show a marked dwarfing. It would seem, therefore, that the question of the effect of crown-gall on peaches and other fruit-trees needs further attention. In certain regions as much as 75 per cent of the trees in nurseries are affected. Referring to extreme cases illustrating the possible economic importance of crown-gall to peaches, it is reported that peach orchards are unprofitable because of the disease. The disease appears to be more easily communicated to the peach under ordinary orchard conditions than to the apple, consequently great precaution should be taken against setting peach-trees in soil where another galled plant has been removed.

For a fuller discussion of the disease, see Apple, page 108.

References

See those listed under Apple, page 112.

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Frost-Injury

Caused by the action of low temperatures

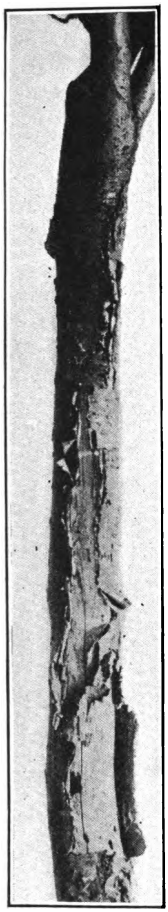

Fig. 79.—Peach-trunk injured by frost.

In New York, New England and elsewhere peaches suffer from the cold. Trees are even killed, a phenomenon which makes the trouble an important one. Trees which are winter-injured show blackened bark in the spring; this injured bark becomes more or less separated from the trunk and the brown wood is evident (Figs. 79 and 80). Such injuries are commonly found at the crown, in which case the disease is called crown-rot or collar-rot (Fig. 79); on the trunk and larger limbs, where it is called frost-canker or sun-scald (Fig. 80); and on the twigs, where it is often referred to as die-back (see also page 300). Toward midsummer frosted trees exhibit foliage which at first is yellow,

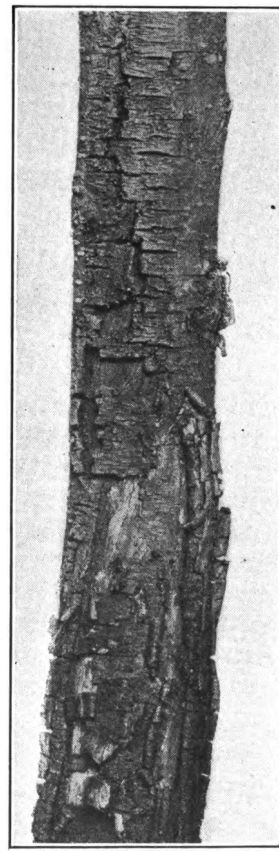

Fig. 80. — Frost-canker on peach-limb.

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then pinkish here and there. If the injured spots are left untreated, bark-beetles and fungi frequently follow the work of the frost. The most common fungus in this connection is *Valsa leucostoma* var. *cincta* (see page 301). In the past, orchards, which probably would have recovered if proper treatment had been given, have been cut down. If the bark clings tightly or is only partly loosened, the trees may recover. Moderate pruning back, removing not more than one-third to one-half of the previous year's growth, good cultivation, and moderate fertilization is regarded as a good course to pursue. See more detailed discussion of frost-injury, its nature and treatment, under Apple, page 35.

Die-Back

Caused by *Valsa leucostoma* Fr. var. *cincta* Rolfs

The first important notice of this disease in the United States dates back about fifteen years. It is a condition more or less common to cherry and peach-trees in old neglected orchards, particularly in central and eastern America. The trouble also prevails in Germany and Australia. A similar disease caused by a closely related variety of the causal fungus affects the plum.

Some varieties of peaches are said to be injured more than others, although none is free from die-back. It is thought that the varying local conditions are responsible for such differences in more cases than can be attributed to varietal susceptibility. Affected twigs and branches are killed back; even large limbs are often severely injured.

Symptoms.

The pathogene attacks only the woody portions of the tree. Rough black cankers, sometimes large affected areas, appear on limbs. This condition is popularly referred to as sun-scald. The lesions center about a bud or a wound. At times there is

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an enlargement at the point of injury. Evidences of the disease are noticeable in late winter or early spring months. Affected twigs are at first purplish, but later scarlet, and leathery. Still later the bark becomes drab-colored, loose and wrinkled. Finally black pustules of the pathogene appear under the bark in these grayish areas. These fruiting bodies break through the bark and become covered with a silvery-white coat, thus dotting the whole diseased portion in a characteristic manner. This condition is often spoken of as silver-twig. Twigs and water sprouts are killed back in mid-winter or later. This killing occurs repeatedly until finally the affected tree is given a ragged appearance. During the growing season an affected limb or twig may be girdled, in consequence of which the foliage assumes a yellowish aspect, then suddenly wilts and dies.

Cause.

While the causal factor is designated as the fungus *Valsa leucostoma* var. *cincta*, the general opinion prevails that other factors such as frost, unfavorable soil, lack of cultivation and other neglect play an important rôle in the production of the die-back disease. Indeed, some prominent authorities hold that the prime cause is the action of low temperatures. The fungus may in any case be regarded as an exciting cause at least, and in its absence the other above-named factors would doubtless exhibit less influence in bringing about the trouble. The pathogene characteristically attacks trees in a weakened condition, like those injured by frost and fire. And this is a real source of danger, inasmuch as such trees when taken at this disadvantage suffer injury far in excess of that induced by frost alone. Furthermore the semi-parasitic nature of the fungus allows it to live indefinitely about the orchard on dead limbs, ready to attack any trees that are in poor condition. A warm spell in the spring followed by a freeze renders the tissue of the peach-limbs favorable to the growth of the fungus.

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In the spring, March and April, conidia coil out in long, reddish brown masses from the fruiting bodies already mentioned. The process is favored by quiet, damp weather, and it takes place in a few hours following the advent of favorable conditions. The spores are scattered by the wind, rain and probably by birds. Within twenty-four to forty-eight hours germination occurs, and the germtube enters the bark through a bud or through a wound of any sort. The germtube soon develops a copious growth of mycelium which is found between the outer bark and the wood in grayish mats. Numerous gum pockets are formed in the cambium and inner bark; many of these unite to form larger ones. The gum thus formed exerts a pressure on the bark, which is ruptured, and an exudation follows. This gum-flow, sometimes referred to as gummosis, is characteristic of the peach and other stone-fruit trees when injured (see page 303).

The fungus may grow down the twigs into the branches, and in this manner large limbs and even trunks become infected. On all affected parts pycnidia are developed in abundance. They occur most abundantly, however, on twigs, while perithecia are most common on the limbs and trunks; the two kinds of fruiting bodies may be intermingled on trunks and larger limbs. Ascospores are discharged from January to April, while conidia, as noted above, are disseminated in March and April.

Sometimes the advance of the fungus is halted, and affected parts may outgrow the disease. Frequently, however, the fungus remains active until late in the fall; its progress is then interrupted by the first freezing weather. With the return of favorable weather the fungus renews activities and the mycelium spreads.

Control of die-back.

The fungus is ever-present on stone-fruit trees, and shows a marked preference for trees already in a poor condition as a

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result of frost-injury and other detrimental factors. It would therefore appear essential that trees be given the best of care; they should be in a mature condition before winter. The control measures involved in frost-injury are discussed on page 43.

Spores of the fungus are formed on all affected parts; these spores cause infections. It is therefore advisable that diseased limbs and other parts be removed. Their destruction would seem necessary on account of the ability of the fungus to live as a saprophyte until such time as the trees are in a condition favorable to attacks by the pathogene.

It has been shown that spraying to protect susceptible parts is not wholly reliable. However, the application of bordeaux mixture and lime-sulfur to the bark greatly reduces the disease, and is believed worthy of further trial. Fall and early spring applications, when no foliage is present, are considered highly desirable where the disease is troublesome. When spraying for leaf-curl, San José scale or other enemies, the bark may be coated for die-back.

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GUMMOSIS

Caused by various factors

The term gummosis, gum-flow, is here used broadly to indicate a sign of disease or injury rather than any specific disease. Like other stone-fruits the peach is subject to a gumming from trunk and branches as the result of almost any kind of injury. The phenomenon has been observed since man began to give attention to stone-fruit trees, and the trouble

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occurs more or less all over the world. Gumming is not confined to stone-fruits, but is known also on citrus trees. Gumming of the former only is considered here.

Symptoms.

Gummosis, or gumming, is usually evident in the spring, particularly after cold rains and when growth is active. Masses of gum, at first glassy or transparent and soft, then amber-colored and hard, exude from the twigs, branches and trunks. Examination will usually show the presence of a break in the bark through which the gum exuded. The gum becomes swollen and sticky in the presence of water. The casual observer will note gum only on the external portions; however, it is formed internally. Sometimes gum is developed internally and there is no evidence of it externally. Gumming often accompanies the blighting of blossoms or twigs, canker-wounds on limbs and trunks and wounds left by pruning operations.

Cause.

The phenomenon of gum-flow, gummosis, is associated with a variety of conditions, and therefore it is difficult on finding gum-exudation to attribute it to any definite cause. Wounds are made inadvertently, and many factors are capable of making them; and wherever a stone-fruit tree is wounded, gum is very certain to exude. But wounds do not always exude gum; an exciting factor is often essential after the wound is made. Just why a tree should exude gum is a matter which has been discussed for years. It is now quite generally held that it is due to some enzyme produced by the host-protoplasm. Some authorities, however, believe that gummosis cannot be due to an enzyme. Others simply say that the gum exudes in response to some stimulus in an effort on the part of the tree to protect a wounded surface, and that the phenomenon represents a retrogressive change in the cell contents. The real causal factor, whatever it may be, passes up the ducts, enters adjoin-

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ing cells, and comes in contact with living protoplasts which are stimulated to release an enzyme; this enzyme causes gelatinization of the primary lamella. This gelatinized gum constitutes the exudate which is forced out through pits into the ducts and adjacent wood cells and fibers. Some of the various causal factors involved in gummosis of the peach follow: (1) frost; (2) fungi, such as *Sclerotinia cinerea, Valsa leucostoma, Coryneum Beijerinckii,* and others; (3) bacteria, such as *Bacterium Pruni;* (4) insects. Among the other factors which have been listed as possible causes of gummosis are: (1) lack of balance between the nutritive processes; (2) galls on the roots; (3) excessive rainfall; (4) severe pruning, particularly when the peach is pruned during the period of its greatest vegetative activity, that is, from April to August; (5) poor cultivation; (6) deep planting; (7) adverse soil conditions; (8) adverse atmospheric conditions; (9) hail; (10) over-bearing. It will be seen that gumming may be due to any one or several of the following classes of factors: (1) climate; (2) adverse soil and nutrition; (3) animal or plant parasites; (4) unfavorable cultural conditions.

Control.

The remedy involved will depend somewhat on the cause. In any case the local affected areas should be cut out wherever this procedure is feasible. In those cases where the exact cause is determined, the measures of control may be found in the discussions given under that heading for the individual diseases concerned (Frost-cankers and gummosis, page 300; gum-flow due to *Sclerotinia cinerea,* page 275; to *Valsa leucostoma* var. *cincta,* page 302; to *Coryneum Beijerinckii,* page 313; to *Bacterium Pruni,* page 310). In general the phenomenon of gumming may be remedied by making conditions favorable to growth of the tree. It is always essential to learn the nature of the exciting factor. (See also Cherry Bacterial-Gummosis, page 181.)

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BLACK-SPOT

Caused by *Bacterium Pruni* E. F. Smith

This peach trouble is known only in the eastern and central United States; so far it has not been reported in any other region of the globe. Not only is its range limited but its history is rather brief, and its importance has prompted scientific writings only during the past decade. But in the short history of black-spot, or shot-hole as it is frequently called, it has assumed a very prominent rôle in the culture of stone-fruits in the humid regions of the South. It is less important in the North and East. In Missouri it is rated as one of the worst of fruit diseases. The losses induced by the black-spot pathogene are not easily reckoned. But in Missouri it has been found that from 1 to 10 per cent of the Elberta peach fruits is injured in well cared for orchards, and from 25 to 75 per cent in poorly managed orchards. All affected fruits are graded second class. Furthermore, such fruits do not ship well, often being completely rotted in transit as a result of rot-fungi which gain entrance through black-spot lesions. The injury is not confined to the fruit alone, but foliage is attacked. Diseased foliage is less efficient than healthy leaves, and so the vitality of the tree suffers. This also results, in the event of early spring infection, in the reduction in size and quality of the fruit. Severe leaf-injury also results in a reduction in the vitality of the fruit-buds and even prevents the formation

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of the normal number, so that the next year's crop of peaches will be materially lightened. Young trees suffer severely; when badly diseased, they become stunted and in some cases are permanently injured.

It should be stated that black-spot affects not only the fruit, foliage and twigs of the peach, but also these same organs of the apricot, nectarine and plum, making four prominent and important stone-fruit trees in the category of hosts for the black-spot pathogene. This fact adds to the economic aspect of this disease. Of the peaches the Elberta is particularly liable to extensive damage. Likewise the Champion, Carman, Alton and others are very susceptible.

Symptoms.

The disease is more likely to be found on young Elberta trees than on peaches of other ages and varieties, although practically all kinds are affected to a varying degree. The leaves (Fig. 81), fruits and twigs show symptoms of the disease.

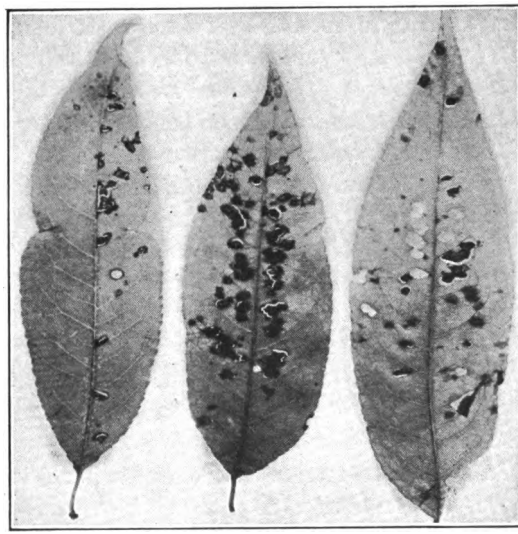

FIG. 81. — Black-spot lesions on peach-leaves. Note the shot-hole effect.

On the leaves a shot-hole effect similar to the leaf-blight of plums and cherries is produced (Fig. 81). The first evidence of the trouble is a leaf-spot; hence the disease is at times called

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bacterial leaf-spot. The spots at the beginning are mere specks, grayish in color, angular in form, and take on a water-soaked aspect. Later, they become brown or purple-brown, or even scarlet, although they are finally dark-brown. Mature spots ordinarily do not measure more than one-fifth of an inch in diameter; many are smaller, but two or more may coalesce so that large areas are involved (Fig. 81). In later stages the affected tissue contracts, dries and falls away, leaving a more or less circular hole (Fig. 81). Several such holes in a leaf give it the appearance of having been riddled with gun-shot, whence the name shot-hole (Fig. 81). Badly diseased leaves fall prematurely; in this way an infected tree may lose from 15 to 75 per cent of its foliage by August. Not all leaves fall at once, but one after another, until finally only a few young leaves remain at the tip of each twig. In this condition the affected tree is characteristic in its appearance.

The lesions on fruits in their early stages are similar to those described for the leaves. Soon, however, the skin is ruptured at affected places, resulting in the production of numerous angular cracks. While the diseased areas are very small — never more than one-tenth of an inch in diameter — they are often so numerous that the crevices run together, thus forming a network of fissures some of which may be an inch in extent. Some growers speak of this condition as bacterial-crack. Black-spot does not usually become evident on the fruit until about the middle of May.

The disease on the twigs shows itself in the form of black spots or cankers; this phase has received the names black-spot, black-tip and bacterial cankers. These lesions are found abundantly in May and June, or even earlier. A single spot develops as follows: surrounding a lenticel there first appears a water-soaked area which bulges out somewhat. As the spot enlarges it elongates, and at maturity extends from one-half to two inches up and down the shoot and from one-half to two-

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thirds of the way around. Older lesions are brownish or purplish brown, then dark-brown, and ultimately are purplish black or jet black and sunken to some extent. Occasionally open cankers form on peach twigs, but, like those on the apricot and nectarine, are much less common than on the plum.

Cause of black-spot.

This is one of the many bacterial diseases which affect plants; the pathogene is known as *Bacterium Pruni.* The causal relationship between *Bact. Pruni* and black-spot or shot-hole has been known nearly as long as has the disease itself.

The black spots or cankers on the twigs are the chief sources of the inoculum in the spring. In other words, the bacteria pass the winter largely on the twigs in the lesions of the previous year's formation. They may also hibernate in the buds in some fashion, and in fallen leaves, if the same are well protected from the sun and air, but these are not important sources of the inoculum in the spring. Rain, dew and insects carry the bacteria from their hibernating quarters to the lower surface of young leaves, in the month of May or later, where they gain entrance through the stomata. The twigs are penetrated by way of the young lenticels. Moisture is essential for this process. Within a week, more or less depending upon the temperature and moisture conditions, and upon the organ attacked, evidences of the disease are visible to the naked eye. In this interim, that is, from the time the peach is entered until the disease is evident, the bacteria multiply rapidly and secrete a solvent which soon breaks down the cell walls, giving the pathogene a good food-supply. In a short time a small cavity is formed beneath the surface of the spot in which many bacteria obtain. These finally escape to the surface, through stomata or crevices, where they lie in sticky masses. They are disseminated to other points, and new infections arise on the current season's twig-growth. With the advent of autumn

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the bacteria become dormant and remain so until spring, when the cycle begins anew.

The weather has a rather marked effect upon the severity of the black-spot disease. Ordinarily an outbreak may not be expected until May; however, if the spring weather is warm and damp, infections appear as early as April 1. A temperature between 68° and 82° Fahrenheit is most favorable to the bacteria. Cold weather not only checks the activities of the bacteria, but also of the insects which disseminate the bacteria. Heavy, driving rains of short duration, even if accompanied by a favorable temperature, are not conducive to the spread of the pathogene; for such rains carry the bacteria to the ground. And should the showers be followed by sunshine and breezes, it is unlikely that a serious outbreak will ensue on account of the fact that the susceptible parts would thus be quickly dried off and the bacteria on the surface would die. Heavy dews, if accompanied by proper temperatures and shaded locations, are favorable to the growth of the bacteria. Slight injury from *Bact. Pruni* results in dry seasons, especially if the spring has been cold.

Control of black-spot.

Reasoning from the facts and phenomena recorded above, treatment may be followed along several lines as tabulated below.

(1) The disease affects the peach, plum, nectarine and apricot. Growers of all four fruits may find the problem of control more complicated than where, for example, only peaches are grown. A few old and comparatively worthless trees of plums, apricots or nectarines may be a constant source of trouble. These should be removed.

(2) The growing of resistant varieties offers little relief at present.

(3) The twigs, fruits and leaves are affected. The injury may be serious on any or all of these organs. The twigs furnish

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hibernating quarters for the bacteria. Careful pruning should include the removal of diseased twigs, in order to destroy the source of the inoculum.

(4) The disease is bacterial, and the general rule that bacterial diseases are not directly controlled by spraying seems to apply to black-spot. It has been found, however, that spraying has an indirect effect in controlling disseminating insects. A special schedule of applications is not warranted.

(5) Orchards which are properly managed, *i.e.* properly pruned, cultivated, fertilized and sprayed, invariably suffer less than those otherwise handled.

(6) The pathogene is spread in nursery stock, consequently in planting new orchards use stock bought only from reliable nurserymen. Younger trees suffer more within five years after setting than do older trees that are in good condition.

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Coryneum-Blight

Caused by *Coryneum Beijerinckii* Oudem.

The disease here discussed is known commonly as California peach-blight, blight, shot-hole, fruit-spot, and sometimes as brown-spot, pustular-spot and winter-blight. The earliest record of it comes from France in 1843. In recent years, particularly from 1905 to 1907, the disease was the subject of considerable comment in California. The first report of it from that State dates back only to 1900, although doubtless the parasite had been there for many years prior to that time. It was first observed in Michigan peach orchards in 1893, and subsequently has been seen in Indiana, Oregon, Ohio and New

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York. The distribution in America would appear rather limited. It occurs widely in Algeria on apricots, peaches and cherries; in France on almonds; in Australia and New Zealand on plums and other stone-fruits; and in Europe commonly on cherries.

This blight disease constitutes a well-known and important factor in peach growing throughout California. Most damage, however, is wrought in the more humid localities, where no variety seems to be immune. Trees are more or less weakened, depending on the severity of the attack, and frequently crops are destroyed. Buds are killed, green fruiting twigs are spotted, and leaves are dwarfed. Premature defoliation is not at all uncommon.

Symptoms.

The effects of the Coryneum-blight are shown early in the spring. This is particularly true under California conditions. The most evident symptoms of blight consist in a gumming and death of the buds on the fruiting wood, accompanied by a splitting of the bark on branches of the current year's growth. Affected buds may die before spring and thus fail to start, or they may start and die later after the young fruit sets. Blighted twigs become spotted, and an exudation of gum ensues; finally death occurs. The spots may show as early as the first of February, at least in California. These are depressed areas in the bark, and are in reality cankers. The leaves may be affected when young. The lesions produced are circular, brownish spots, with dark-red margins; finally the affected tissue falls away, leaving a hole in the leaf where each dead spot first occurred. Where several such holes occur there results a shot-hole appearance. This shot-holing is more common as a result of the first lesions produced; lesions developed later in the season do not exhibit this peculiarity. On the latter spots small grayish tufts of the pathogene may be seen. Lesions produced on peach-fruits resemble somewhat

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the effect induced by the San José scale (Fig. 82). At first the spots are small and purplish red. As the diseased portion enlarges, a light-colored area develops in the center (Fig. 82). Later the spots are brown and confluent. In extreme cases the fruit becomes cracked and a flow of gum follows.

Cause.

The lesions described above are induced by the fungus *Coryneum Beijerinckii.* The chief period of infection appears to be from December to February; most inoculations occur about January first. In California no infections occur after the end of April; in Oregon these take place from May 10 to June 15. Rainy weather favors infection.

FIG. 82. — Coryneum-blight lesions on peaches.

The conidia of the fungus are carried to the buds, leaves and fruits, germination occurring in a short time in the presence of moisture. After infection, spore-cushions break through the skin of the various parts affected. These are visible to the naked eye as dark spots, and are most abundant about leaf scars and roughened portions of the bark on defoliated twigs in the spring and summer. They also develop on leaf-spots. The conidia appear to live through the summer lodged in the bud-scales, being very resistant to desiccation.

Control.

It has been found in the Pacific Coast regions that Coryneum-blight can be controlled by fall and spring spraying. Peaches should be sprayed about November first, just as soon as possible after the fruit is harvested. Bordeaux mixture 6–6–50 is

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regarded as the best fungicide available at present. This autumn application alone has been effective under California conditions, and it may also prove entirely satisfactory for Oregon orchards. The present recommendations, however, for Oregon conditions include supplementary sprayings in the spring, using self-boiled lime-sulfur 8–8–50. Two applications are made as follows: (1) May tenth; (2) June first; if the disease is unusually prevalent and the weather rainy, the second spring application may be made about May 20 or 25, and a third spraying about June 5 or 10.

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POWDERY-MILDEW

Caused by *Sphærotheca pannosa* (Fries) Lév. var. *Persicæ* Woronichine

It was formerly believed that the peach and rose mildews were the same, but recently it has been shown that the two are not absolutely identical. The peach powdery-mildew probably occurs also on the nectarine.

This disease is undoubtedly world-wide in its range, and has been found occasionally in the United States since 1886. It affected peach trees in that section of the country about Maryland and Delaware from 1886 to 1891, occurring annually during those years. In 1891 the disease was observed in Michigan and Georgia, and three years later, 1894, it was reported as causing serious damage in western New York. As a rule, however, this disease is of relatively little consequence in New

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York State. In 1905 peach-mildew attracted attention in Colorado, and in 1907 it was discovered in Nebraska and Utah.

While peach-mildew is not everywhere destructive, it does cause considerable damage at times. Twigs may be checked in their growth or even killed; the foliage may be greatly reduced in size and efficiency; and the future health of the tree may be impaired. Seedlings are often badly affected, and nursery stock suffers more than orchard trees. The last, however, are not uncommonly affected, which, in case of severe infection, may be entirely ruined for market. Fortunately, not many trees in a given orchard are extensively damaged.

The susceptibility of varieties is not marked; this is true at least for Colorado conditions. In Michigan the popular opinion prevails that only those varieties with serrate leaves and lacking glands are affected with powdery-mildew. It has been found by authorities, however, that this is not always true. In Ohio, it has been observed that the Early Crawford may be badly affected. At Geneva, New York, the disease was found on but eight out of three hundred and fifty varieties: Bailey, Conkling, Tillotson, Simmons No. 1, Wright, Morrell, Thomas Rivers and Illinois Peach. In western New York, the Crawford and especially the Denton are most susceptible.

Symptoms.

Injury is done by the mildew-pathogene to the leaves (Fig. 83), twigs (Fig. 83) and fruits (Fig. 84). Perhaps the fruits are least affected of all. Along the lower surface of the foliage, particularly at the midrib, are to be found abundant, irregular, white blotches — the mildew (Fig. 83). It may also occur on the upper surface. As a result of the affection the foliar parts crumple and curl, the edges rolling toward and parallel to the midrib (Fig. 83). Young leaves, when affected, fall prematurely. Older leaves are dwarfed. Signs of the disease may first be observed in early summer.

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Only young growing twigs are affected. Conspicuous white patches appear on the surface. The bark becomes dry and brown at infected points, and in severe cases the leaves fall as a result. Finally the bark shrivels and the young tips become curved; growth is thus checked and the twigs may die.

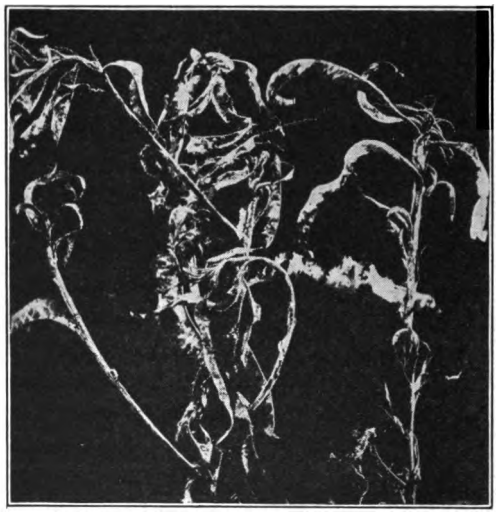

FIG. 83. — Peach-mildew on shoots and leaves.

The disease may involve the whole or, more commonly, only a portion of the fruit. It appears as a frost-like covering which assumes a pure white aspect due to the presence of the mildew-pathogene (Fig. 84). The affected peach flesh hardens and the skin turns brown, and finally the peach cracks. Young fruits are often caused to fall prematurely.

In Oregon two types of symptoms have been noted: (1) mildew scattered on the fruit and leaves, twigs less affected; (2) general on all parts. The former is thought to be the true peach-mildew disease; the latter the cherry-mildew (see page 177).

Cause.

The name of this powdery-mildew organism is *Sphærotheca pannosa* var. *Persicæ*. Its mycelium grows in dense superficial patches, giving to the lesions the powdery-mildew aspect already described. Soon after the fungus appears, great numbers

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of conidia are developed. From the mycelium erect stalks are formed which bear conidia in chains at the tip. These stalks are the conidiophores; they, with their conidia, add to the felt-like, whitish growth, covering the diseased portion. The conidia are scattered throughout the summer, and those which fall on peaches will, under favorable conditions, germinate to start a new mildew spot. From the germtube a dense mat of mycelium develops. At intervals over the hyphal system, sucker-like bodies, called haustoria, are sent into the epidermal

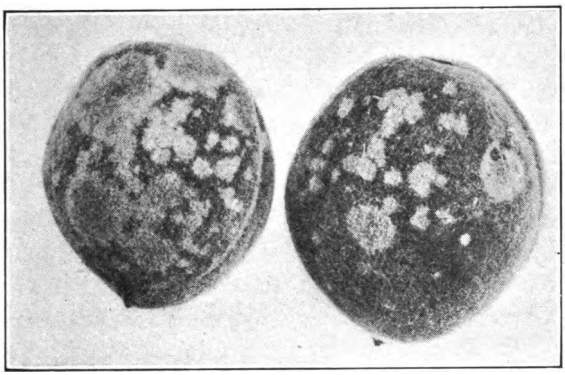

FIG. 84. — Peach-mildew on fruits.

cells. These haustoria are the feeding organs of the fungus, and as a result of their activity the attacked portion becomes brown and cracked.

After midsummer perithecia may be developed. These bodies when formed carry the fungus through the winter, but as a rule they are extremely rare. When found they occur more on twigs than on leaves. In those cases where perithecia do not develop, it is thought that the fungus hibernates as mycelium in the buds.

The fungus thrives best in a warm, moist, shaded location. Trees that are closely planted are more likely to be affected;

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this is due to the increased moisture content of the air about such trees.

Control.

Wherever possible those conditions which favor the fungus should be avoided. Attention should be given to trees planted near one another, and to those with a dense foliage. A good circulation of air and plenty of sunlight is essential in this connection.

As a fungicide, copper salts have not been generally effective. On the other hand, sulfur has proved to be satisfactory. Sulfur applied either as flowers of sulfur (dust) or as lime-sulfur solution is a success. Lime-sulfur 1 to 50, with the addition of three pounds of iron-sulfate to fifty gallons of the mixture to increase the fungicidal properties and adhesiveness, is efficient. Sulfur dust is desirable in that less time is required for the application. This should be applied early in the morning while the foliage is still damp. If the weather subsequent to the dusting operation is warm and dry, the fungicide may be expected to be more efficient. Rainy periods, on the other hand, tend to decrease the efficiency by washing the sulfur away; however, this is not serious except in cases of rain periods of long duration. The fungicide used for powdery-mildew should be applied as soon as the disease appears. The weather and consequent abundance of the disease determine the number of later applications. In some years two applications are sufficient; in others it is profitable to give the trees five or six treatments at intervals of about one or two weeks. In the far West the spraying schedule for the Coryneum-blight and brown-rot is satisfactory for the scattered form of powdery-mildew. When the generalized form, that is on all parts, is present (that caused by the cherry powdery-mildew pathogene, *Podosphæra Oxyacanthæ*), the control problem is more difficult. In any case it is best to remove badly diseased seedlings.

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ARMILLARIA ROOT-ROT

Caused by *Armillaria mellea* (Fries) Quel.

In some sections of the country the peach among many other fruit-trees is injured by the Armillaria root-rot. For a fuller discussion of the disease, see Apple, page 96.

CLITOCYBE ROOT-ROT

Caused by *Clitocybe parasitica* Wilcox

The disease in question has been called rhizomorphic root-rot. It occurs also on the apple and cherry. Facts concerning its range, symptoms, cause and control are discussed under Apple, page 102.

RUST

Caused by *Puccinia Pruni-spinosæ* Pers.

This rust disease, due to *Puccinia Pruni-spinosæ*, occurs on practically all the stone-fruits, but in the United States it affects chiefly the plum. Certain stages in the development of

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the rust fungus are found on hepatica, anemone and thalictrum, all common wild flowers. The trouble shows on the lower surface of the leaves of stone-fruits as small, round, powdery, yellowish brown pustules. Opposite these pustules, on the upper surface, the leaf turns reddish yellow in local areas. Such lesions frequently become limited and the affected tissue falls away, leaving a shot-hole effect in the leaf. These spots and holes may be confused with those caused by the California peach-blight fungus (*Coryneum Beijerinckii* Oud.), but are distinguished from the latter by their smaller size. Peach rust has a rather general geographical range, but does not ordinarily do damage sufficient to warrant treatment. See also Plum, page 377, and Apricot, page 156.

Silver-Leaf

Caused by *Stereum purpureum* Fries

While this disease affects primarily the plum and apple, many other fruit-trees and shrubs are liable to it. The peach is sometimes victim to silver-leaf, but it never suffers so generally and so extensively as others of the stone-fruits like the plum. The characteristics, cause and other features are discussed under Plum, page 368.

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(See additional literature listed under Plum, page 373.)

Frosty-Mildew

Caused by *Cercosporella Persicæ* Sacc.

In the central Atlantic states there occurs a peculiar disease of peach-leaves. It is more common from Maryland south-

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ward, being found in North Carolina, Virginia, West Virginia and Arkansas. It also occurs, but rarely, in the state of Ohio. The disease is not usually serious, although it prevails to a very noticeable extent in damp, shaded localities on trees bearing dense foliage. It was unusually prevalent on the Delaware and Chesapeake peninsula in the fall of 1891. Pale, yellowish or olivaceous spots with definite margins are produced by the fungus *Cercosporella Persicæ* on the upper surface of the leaves; on the lower surface, opposite the yellowish spots, will be found a delicate frost-like growth of the fungus—the conidiophores.

Little is known of the causal fungus and its control.

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Stem-Canker

Caused by *Phoma Persicæ* Sacc.

In 1896 a peculiar stem-trouble was noted in Ohio affecting primarily nursery-stock and the twigs of older orchard trees. Two years later it was described, and was called constriction disease, or stem-blight. It has subsequently been reported from Connecticut and New York on seedling peaches. The affected twigs are partially or completely girdled by a cankerous formation. The parts above the lesion do not die immediately, but eventually they succumb, as evidenced by a yellowing and a premature defoliation. Just above the canker a swelling of the stem occurs, resulting in a constricted appearance. Scattered over the canker will be found numerous pycnidia of the fungus which show as black specks. The pathogene develops

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best on trees in a weakened condition, and for that reason the careful grower may expect that the trouble will never be of great consequence.

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CHAPTER XI

PEAR DISEASES

THE most injurious diseases affecting the pear are fire-blight (page 323) and scab (page 332). Fire-blight is commonly found on the apple and quince, and on these three fruits great damage may be wrought. Pear-scab is peculiar to that fruit; it is not the same disease as apple-scab, in spite of the popular notion which prevails in the affirmative. Less prominent diseases, like pink-rot, superficial bark-canker and leaf-blight, are also found on the apple and quince.

FIRE-BLIGHT

Caused by *Bacillus amylovorus* (Burr.) Trev.

This disease is known to affect several fruit-trees and a few ornamentals belonging to the apple family. The discussion presented here concerns only the pear.

All varieties of the pear are more or less susceptible to fire-blight. But it is generally believed that the Bartlett, Flemish and Clapp Favorite are more frequently and more severely attacked than the Kieffer, Angoulême and Seckel. However, these apparent variations in resistance may not be traced to an inherent difference among kinds of pears, but to other factors and conditions frequently overlooked. The presence of the disseminating agents (insects), the source of the inoculum, and the period of growth-activity of the host rule the situation more than real immunity.

The disease is very generally known as fire-blight. It is also

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referred to as pear-blight, blossom-blight, twig-blight, fruit-blight and blight-canker. Various combinations of these names are used. The term blight signifies a sudden killing of a part or all of the plant, and in case of fire-blight the blossoms, leaves and limbs are so affected.

The disease is of American origin, having first been known and described in 1794 from the Hudson River Highlands in New York State. Since its discovery fire-blight has appeared in the South and West, and about 1880 it was the chief topic of discussion in the horticultural meetings in the Middle West. For several years it was unknown west of the Rocky Mountains, but about 1900 it was described from California. The disease now has a very general range throughout the United States and southern Canada, but does not occur elsewhere.

Fire-blight is the most universally destructive of all pomaceous fruit diseases. The range and perennial nature of the host and the nature of the disease account for this condition. Being an epiphytotic disease, it may appear suddenly in a locality and rapidly cause severe injury or complete destruction to the pear industry of that section. Nursery-stock suffers severely, and often thousands of affected trees are ruined. In some cases entire blocks of pears are destroyed. Orchard trees may be killed in one season. In some years the attack subsides, but with the recurrence of favorable conditions the ravages are renewed. The disease is said to be generally least troublesome in the United States and in portions of Canada bordering the Great Lakes. But in New York and Michigan the outlook is sometimes discouraging. In the region south of these two states Bartlett pear-growing has been largely abandoned. In the southern states the Kieffer, generally regarded as resistant to the disease, was once widely grown, but its culture has been discontinued, owing to the destructiveness of fire-blight. In California two-thirds of the Bartlett trees had been destroyed by 1908.

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The nature of the injury makes it difficult to obtain accurate figures representing the annual loss from this disease, either in a given locality or in the country as a whole. The blossoms are affected, which means a loss of the current year's fruit-crop, and often that of the next on account of the death of the spur. Twigs may blight in great numbers, thus destroying the twig-growth of the current year; this may later result in death of the larger limbs bearing such twigs. Cankers which may eventually kill the tree are produced on the limbs. Finally, the fruit may be attacked directly; in such cases it is rendered unmarketable.

FIG. 85. — Fire-blight; the bacteria have spread from the blossom into a twig, forming a canker.

Symptoms.

Attacks on the blossoms result in blossom-blight (Fig. 85). This phase of the disease is evidenced by a sudden wilting and darkening of the young fruits after the petals fall, followed by similar changes in the spur. This is a very common form of the disease in New York State. The most striking symptom to be recognized by the grower is that of twig-blight (Fig. 86). In the Middle West twig-blight is the most common form of blight. The affected organ is blackened and bears darkened, drooping leaves, the whole appearing as if burned by fire. In no other diseased condition of fruit trees does the foliage cling so tenaciously to the dead branches. Cankers occur in the bark of the body or of large branches (Figs. 87 and 88). In the winter the spot is dark, smooth and sunken, the margin definite and

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usually marked by a crevice (Figs. 87 and 88). In the spring the canker has a soaked appearance, the advancing margin being indefinite or raised. Sometimes milky or reddish brown

FIG. 86. — Twig-blight. Tip of a shoot blighted by the fire-blight bacteria.

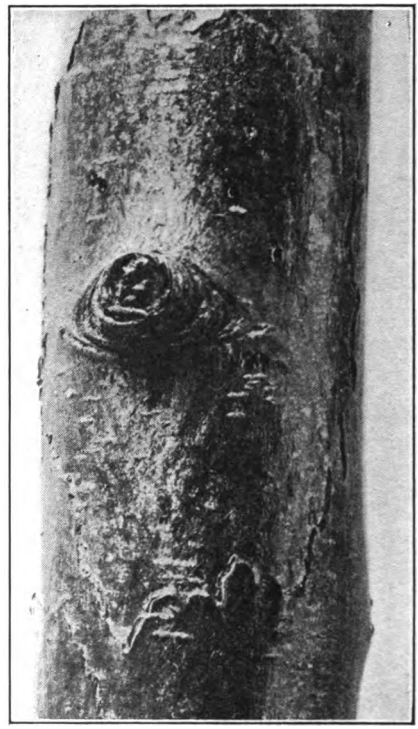

FIG. 87. — Hold-over canker. Note that it surrounds a stub of a twig.

drops ooze from the lenticels (Fig. 89); the presence of this ooze, however, is not a constant character. Not infrequently fire-blight and winter-injury are confused. In contrast to a sudden and local dying of the affected organs in the case of fire-blight, winter-killing manifests itself by a general wilting

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and a uniform browning of all the foliage simultaneously. Cankers most frequently surround the base of a spur (Fig. 85), watersprout, or small limb (Figs. 87 and 88).

Cause.

Fire-blight is caused by the bacterial pathogene *Bacillus amylovorus*. This pathogene passes its entire life-history within the tissues of the living host, except, of course, during dissemination from one place to another. It cannot survive long, even in the dead parts of the plant attacked. The organism passes the winter in an inactive condition in the tissues along the margin of the blight-cankers both on larger limbs and on twigs; such lesions are sometimes called hold-over cankers (Figs. 87 and 88). In the spring the bacteria become active, multiply rapidly, and spread into adjoining healthy tissues. Great numbers of them ooze forth in sticky masses from the lenticels (Fig. 89). This ooze is the source of trouble for the season; it is visited by wasps, bees, flies, beetles, bugs, aphids, curculios and leaf-hoppers, any or all of which insects may carry the bacteria to the opening blossoms, to the tender growing-tips of twigs, or to wounds in the bark. Pruning tools are also agents in transmitting the bacteria. In these various in-

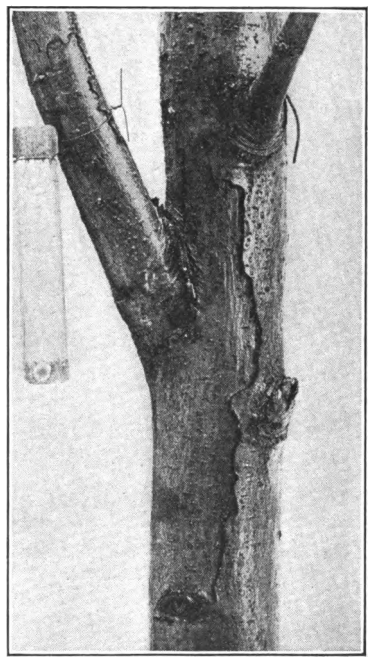

FIG. 88. — Hold-over canker.

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fection-courts the bacteria multiply rapidly and within a few days infection results. The first evidence of the disease in the spring is blossom-blight, the tender tissues of the blossom and of the embryo fruit being killed suddenly. The bacteria in a blighting blossom are carried from it to other blossoms by insects, especially bees, and so the pathogene spreads through the orchard, and from one orchard to another.

FIG. 89. — Oozing fire-blight canker. Note drops of exudate.

From the blossoms the bacteria work their way down the pedicel to the spur, killing the bark and causing the leaves as well as the blossoms to wither. The leaf-tissues are not usually invaded. From the spur the bacteria may pass to the other healthy pedicels of the cluster and finally enter the fruit through its base. Fruit-blight may also arise by the bacteria being deposited in wounds made by the curculio and other insects (Figs. 90 and 91). The bacteria are similarly introduced into the growing tips of twigs and watersprouts by aphids, twig-blight resulting. Affected twigs emit the ooze which serves as a source of inoculum for other twigs, shoots and blossoms. In most varieties of pears, twig-infection, unless removed, is inclined to continue down the main limb and even into the body of the tree. Watersprouts frequently mark the center of a canker, indicating the manner of entrance of the bacteria.

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Suckers which arise from the crown, at or below the surface of the soil, are often blighted, allowing the bacteria entrance into the bark of the roots. Trees may die from such a form of attack. Grafts are especially disposed to blight during the first year or so on account of their rapid and succulent growth. Wounds in the larger limbs or the body of the tree may serve as centers of cankers. Here the bacteria are carried by the bark-boring beetle and deposited in their borings. In these cankers and blighted limbs and twigs the bacteria pass the winter. With the return of the warm weather and rains of the spring the rise of sap encourages the growth and multiplication of the bacteria, which ooze out and afford the source of the inoculum for the opening blossoms.

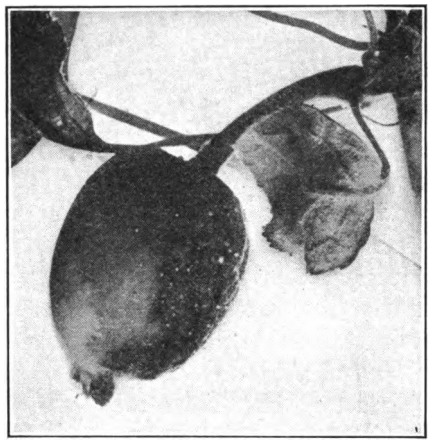

FIG. 90. — Fire-blight on apple fruit; drops of bacterial ooze on the surface.

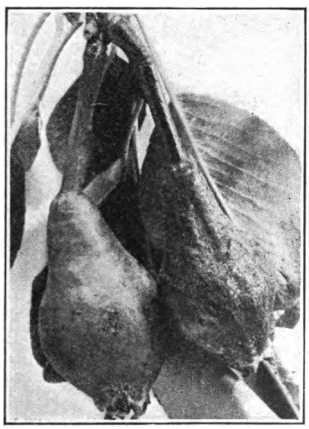

FIG. 91. — Fire-blight on pear fruit; healthy fruit left, diseased fruit right.

Weather conditions should not be confused with the causal factor of fire-blight. On the other hand, the weather is correlated to some extent with epiphytotics of the trouble. Late frosts may stop blight by killing certain of the dis-

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seminating agents, such as aphids. Should no frost occur and were the spring backward, the insects would multiply rapidly while blossoming would be retarded, hence at blossoming large numbers of insects are at hand to disseminate the bacteria rapidly. During the growing-period, immediately following blossoming, should the weather become hot and dry, the rapid growth is checked and the otherwise succulent tips become harder and more woody. Such shoots are less liable to blight-infection. On the contrary, muggy periods favor the disease in that the tissues become gorged with sap and thus offer less resistance to the invading bacteria.

Control of fire-blight.

In attempting to control fire-blight, the following important points should be borne in mind: (1) that the disease is caused by bacteria which gain entrance to the host tissues only through wounds, or punctures by insects, into succulent, rapidly growing tissues, or through the nectaries of the blossoms. (2) That insects of several kinds are the usual agents of inoculation. (3) That practically all pome fruit-growing sections in North America are infested, and therefore there is always a source from which the bacteria may be disseminated. (4) That all known varieties of the hosts, on which the blight-organism occurs, are more or less susceptible; while some show resistance, none are wholly immune. Therefore control consists chiefly in the elimination of the pathogene from the infected trees. This is accomplished by a strict application of the following operations: (*a*) inspect all pear trees in the autumn and again in the early spring before the blossoms open, and cut out and treat all cankers in the body and main limbs. With a sharp knife, or draw-shave, remove all the diseased tissue, wash the wound with corrosive sublimate (one tablet to one pint of water), and, when dry, paint the wound with coal-tar or lead paint, preferably the former. The wound-dressing will need renewal every year or so. (*b*) Throughout the summer,

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beginning with the fall of blossoms, make an inspection every few days of the young trees. Break out the blighted spurs and cut out diseased twigs, making the cut at least six inches below the diseased portion. Disinfect the cuts with corrosive sublimate. (*c*) Remove all watersprouts from the trees two or three times during the season. (*d*) In the nursery remove the blossom-buds, particularly of the quinces. Here inspection must be frequent, particularly in susceptible stock, in order to keep the disease under control. It is often necessary to inspect certain blocks daily, the diseased twigs being cut out as soon as observed. When budded stock of the first year becomes affected, the trees should be dug out, since cutting below the diseased area causes the trunk of the young tree to be crooked and therefore not marketable. (*e*) Control the insects; those which are active in disseminating the blight bacteria have been enumerated (page 327). The real point of attack lies in this phase of the problem. (For discussions of fire-blight on other fruits, see Apple, page 21; Apricot, page 159; Cherry, page 191; Plum, page 386; and Quince, page 387.)

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Scab

Caused by *Venturia Pyrina* Aderh.

Although apple- and pear-scab are very similar in all respects, they are not the same disease. The general opinion prevails among growers that because pear-scab looks like apple-scab, and because the two diseases bear similar names and are controlled in approximately the same way, the one is identical with the other. But the causal fungus in the one case is specifically different from that in the other, and therefore a scabby pear-tree is in no way dangerous to an apple tree and vice versa.

Pear-scab is perhaps as well known to growers of this fruit as any other disease except fire-blight. It has a wide range, but is controlled with less difficulty than fire-blight, and consequently is not nearly so greatly feared. On the other hand, it does considerable damage. For example, it is estimated that pear-growers in the State of Ohio lost in 1905 at least $50,000 through this disease alone. In practically all regions where it occurs it is second to fire-blight in importance. In California losses are put at 25 to 100 per cent in unsprayed orchards. In all carefully sprayed orchards the losses are much less, and often not appreciable. Damage is wrought in nurseries as well as orchards.

The quality of the fruit, rather than the quantity, is affected. The taste is not altered noticeably, but the appearance and keeping qualities, as well as canning and drying qualities, are considerably impaired. Affected fruit sometimes falls and is then worthless. When blossom-pedicels are affected, a loss of the set of fruit results. This happens frequently.

Not all varieties of pears suffer alike. Scab is notably common on the Flemish Beauty, Winter Nelis and Easter Beurré. The Duchess, Seckel and Summer Doyenne are among the other susceptible varieties. Many others are at times scabby but not so frequently nor to such a marked extent as in the case

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of those varieties already mentioned. The Kieffer shows considerable resistance in some parts of the country.

Pear-scab occurs practically everywhere the fruit is grown. It was found in Belgium in 1832, but was not reported as doing great damage until 1875. Elsewhere in Europe scab prevails commonly on the pear. In the United States outbreaks occur in New England, the Middle West and along the Pacific Coast. It was particularly destructive in 1906 over the country generally; in 1898 it was epiphytotic in Ohio; in 1902, 1910, 1912 and 1915 it was unusually prevalent in New York; while it prevailed extensively in California in 1904, 1905 and 1906.

Symptoms.

The disease is found on the fruit, leaves and twigs. On the fruit (Fig. 92) the spots are at first olivaceous, velvety and

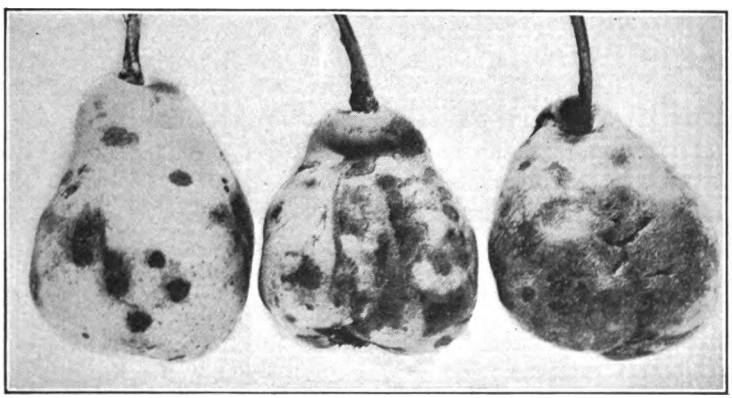

FIG. 92. — Pear-scab.

circular in form. Later in the season this velvety aspect becomes corky and the skin is cracked, sometimes in a T-shape fashion (Fig. 92). At times growth of the young fruit is halted, and so when such pears are mature they are considerably distorted. Affected fruit may drop when the size of a cherry.

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Lesions also occur on the fruit-pedicels. On the leaves the disease exhibits itself as spots very much like those on the fruit. Lesions on the foliage are abundant and conspicuous on the lower surface. This is particularly true on certain varieties, as, for example, Winter Nelis. Pear-scab is common on the twigs. In this respect the disease displays a difference from apple-scab; the latter affects twigs rarely in comparison to pear-scab. Young twigs are affected chiefly, but the lesions are not so conspicuous as on the fruit and leaves. The spots suggest the appearance of a scale insect. On one- and two-year-old twigs the affected areas are velvety and enduring, whereas those on older twigs are soon lost to view, the bark being sloughed off and replaced by healthy tissue. Not infrequently the blossom stalks are affected, in which case a dark-brown spot is produced and the young fruit falls; thus the young fruit fails to set.

Cause.

The scab spots already described are composed chiefly of mycelium of the fungus *Venturia Pyrina.* Numerous radiating and branching hyphæ make up the velvety layer visible to the naked eye. Before the fungus completes its growth on the various organs attacked, dense erect conidiophores arise from the mycelium. Within a short time after their development numerous conidia are formed. These spores are carried to other susceptible organs of the pear where new spots are initiated. Spores which by any chance fall on the apple do not produce the disease. Those which fall on the pear begin their development by germinating. Mycelium is soon formed and a scab spot is perceptible. If a young fruit be attacked, the cells beneath the lesion cease growth, as evidenced by the dwarfing of the fruit. This indicates strongly that there is a drain on the cells in the infected region. The fruit reacts against the fungus by forming cork, which is most evident in old scab spots where the fungus has ceased vigorous develop-

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ment. However, much of the food for the fungus no doubt comes from the attacked cuticle.

Infection of the various parts continues throughout the growing season. In the fall the affected leaves drop to the ground, carrying the fungus with them. Soon the mycelium changes its superficial habit and permeates the entire leaf. Before cold weather, perithecia begin their development within the old dead leaf-tissues. These bodies remain immature until spring, when they resume growth. This begins at least by the time the pear-tree starts into growth. By the time the blossom-buds are showing white the perithecia contain mature ascospores which are discharged during periods of moisture. Their ejection is accomplished with force enough to carry them into the air, where they are easily caught by the wind and are blown to the opening buds. The germination of these spores finally results in infection. The period over which ascospores are discharged is not definitely known, but reasoning from the known facts concerning the apple-scab fungus the period probably extends over several days.

The pear-scab fungus also passes the winter on the twigs. The mycelium and conidia remain alive from autumn until spring, when new infections are initiated. Twigs are commonly affected, so that this method of hibernation is unquestionably of considerable importance. It is not known nor believed to be true that the fungus winters over on fallen fruit.

Control.

Pear-scab, although found everywhere, can be controlled effectively. That it is not held in check in many orchards where spraying for it is done is no indication that it cannot be prevented. Time and thoroughness of the applications of fungicides are of prime importance.

The number of sprayings will depend somewhat on the history of the management of the orchard in the past. If the orchard has been well sprayed, three applications will suffice

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under ordinary conditions. If spraying has been neglected in the past, then more than three applications will be necessary on account of the constant source of trouble on the twigs. A dormant spraying is advised for the far West. This is done late in the winter to kill the fungus on the twigs. A second dormant spraying is recommended in California, to be made just before the buds swell. Other applications follow: (1) a few days before the blossoms open; blossom-buds exposed and separated; (2) when the blossoms have for the most part fallen; (3) two weeks after the second application. In the East the two dormant sprayings advised for California conditions are not practiced for scab, although many pear orchards are sprayed just before the buds swell for blister-mite. The value of this application for scab needs investigation. Lime-sulfur 1–50, or bordeaux mixture 3–3–50, may be used; the latter is much less desirable on account of the russeting which it produces on the fruit. Since the fungus hibernates in the twigs and fallen leaves, it is advised that in pruning and cultivation attention be given respectively to the removal of badly infected twigs and to the burial of old leaves.

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Pink-Rot

Caused by *Cephalothecium roseum* (Fries) Cda.

This is one of the most common of the storage rots of the pear. It also occurs while the fruit is still on the tree. Where found it may be seen on examination that the pink-rot pathogene, *Cephalothecium roseum*, has gained entrance through scab spots, or through breaks in the skin due to careless handling of the fruit. When scab is controlled and the fruit carefully handled, pink-rot is reduced to a minimum. (See Apple, page 117.)

Mycosphærella Leaf-Spot

Caused by *Mycosphærella sentina* (Fr.) Schröt.

The pear-leaf disease here discussed is better known as leaf-spot, Septoria leaf-spot and ashy leaf-spot. It was first described in the United States in 1897. During that and the succeeding year it was a prominent and important pear trouble. In fact, the disease is usually common every year. The ravages of the disease have always been confined to the eastern portion of the United States, whereas in Europe it is widespread. Orchard trees and nursery-stock suffer alike. A few cases have been found on apple-foliage.

This leaf disease is capable of bringing about considerable damage to the pear. Leaves are frequently so badly infected that premature defoliation takes place in August or earlier. This results in checking the growth of the affected tree and prevents the proper ripening of the buds and shoots. This untimely defoliation may also cause the tree to put out new growth which cannot harden before winter. Thus the tree is made sensitive to winter-injury.

Some varieties of pears are more affected than others. It

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was observed several years ago that the Bosc, Sheldon, Seckel, Anjou, Bartlett and others are injured more than the Flemish, Clairgeau and Duchess. The Kieffer, Lawrence and Mount Vernon are relatively resistant to leaf-spot. In the nursery, budded pear-stock, after the first year, may show occasional lesions, but budded stock of two or more years is often badly injured late in the summer.

Symptoms.

Only the leaves are affected. The mature spots are recognized by their well-defined angular margins and their grayish white centers in which a few pycnidia develop (Fig. 93). They are found on the upper surface. The formation of a spot proceeds as follows. The affected tissue becomes brownish, which discoloration enlarges until the mature size of the spot is reached. These usually measure about one-sixth of an inch or less in diameter. The margin, while sharply defined, is angular and crenulate. With age the brown color at the center turns grayish white and becomes somewhat transparent. About this light-colored central area is a brown zone which merges into a purplish zone and finally into the healthy green portion of the leaf. Ordinarily lesions are not abundant, but under exceptionally favorable conditions numerous spots develop over the leaf-surface. Several may merge, involving large areas of leaf-tissue.

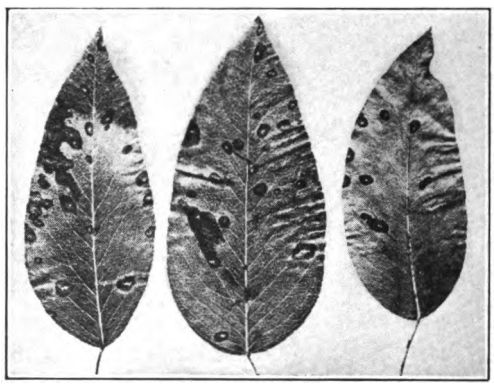

FIG. 93. — Mycosphærella leaf-spot on pear-leaves.

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Cause.

The causal organism is the fungus *Mycosphærella sentina;* it is better known as *Septoria Pyricola,* although the former name is now accepted.

The fungus passes the winter in fallen leaves which were diseased the previous summer. From these leaves ascospores are discharged into the air, and they are then carried to the new leaves. Within about fifteen days spots will become visible to the naked eye. During this period the fungus spores have germinated, the germtubes have penetrated the leaf, and the developing mycelium has established a food relation with the pear leaf-cells. The attacked cells are killed, and in a few days after the lesion becomes evident summer fruiting bodies begin to appear. They are formed beneath the upper surface of the leaf. They are composed of interwoven hyphal threads and break through the leaf-surface at maturity. When thus exposed they appear as black, flask-shaped bodies and are easily seen with a lens or the naked eye. Within two weeks after the effects of the fungus on the leaf begin to show, countless mature pycnospores are developed within the pycnidia. When the fruiting structure is moistened, these spores coil out through the mouth of the pycnidium and are readily disseminated by the rain. They fall on other leaves and new infections result. This repetition continues throughout the summer. In the autumn the leaves fall to the ground, and those affected by the leaf-spot fungus carry the mycelium with them in their tissues. In the fallen leaves the fungus becomes saprophytic, living throughout the winter on the dead tissues. With the advent of spring perithecia are formed, within which ascospores are matured for the first infections. It is held by some authorities that the pycnospores may winter over. The evidence on this point is as yet incomplete.

Control.

Neglected trees are worst affected. It has been found that

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spraying the pear foliage with bordeaux mixture 4–5–50 or lime-sulfur solution 1–50 will satisfactorily control Mycosphærella leaf-spot. Three applications are sufficient in the orchard; (1) just after the petals fall; (2) two weeks later; (3) two weeks after the second application. In the nursery it is recommended that two-year-old budded pears be sprayed shortly after the new leaves open. Subsequent sprayings should be made at two-week intervals; three sprayings in all will prove profitable for two-year-old budded stock. But since one-year-old budded stock is rarely attacked to a damaging extent, it is regarded as unprofitable to spray such trees for this disease.

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SUPERFICIAL BARK-CANKER

Caused by *Myxosporium corticolum* Edg.

A roughening of the bark of both pears and apples is exceedingly common in eastern United States. (See Apple, page 130.)

FROST-INJURY

Caused by the action of low temperatures

At times pear trees are severely damaged by frost. The trees are often planted on low, rich soil, and from their location are more subject to cold. A difference in elevation of

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only a few feet may often be sufficient to determine whether the trees will be severely affected or not. (For a fuller discussion of the question of frost-injury, see under Apple, page 35.)

Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

The pear, like most other fruits, is affected with galls both at the crown and at the tips of the roots. While orchard trees show the enlargements, there is a greater tendency for nursery stock to be affected. In any case the destruction to pears is less rapid, and therefore less extensive, than to peaches or apples. Hairy-root, another form of the crown-gall disease, is less common on the pear than on other fruit-trees. Both the galls and hairy-roots may be induced by factors other than *Bacterium tumefaciens;* among such agencies may be noted (*a*) improper wrapping of grafts, (*b*) heavy applications of nitrogenous fertilizers, and (*c*) the woolly aphis. A discussion of the galls, the causal factor, and remedial measures are treated more fully under Apple, page 108.

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Eastern Rust

Caused by *Gymnosporangium globosum* Farlow

Pear rusts occur generally throughout the world. In the United States two important rust diseases of pear exist, but

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fortunately for all concerned, the one is confined to the eastern part of the country, while the other (see page 345) occurs only on the Pacific Coast. They are accordingly named eastern rust and Pacific Coast rust.

The rust disease here considered is very similar to the apple-rust (see page 63). As already indicated, this trouble has a wide range over the United States east of the Mississippi River. It has been recorded from Connecticut, Massachusetts, New Jersey, North Carolina, Alabama, Mississippi, Indiana, Wisconsin and Iowa. It is relatively rare in New York State.

Rust is destructive in that it interferes with the normal activities of the plant. In severe cases defoliation results. Affected fruits are rendered worthless, and are usually less than one-half normal size. Several Japanese varieties of pears are likely to be injured. The Worden, Bartlett, Bosc and Duchess are said to remain relatively free from the disease.

Symptoms.

Leaves and fruits (Fig. 94) are rusted. In June, orange-colored or dark-brown spots with red borders appear on the upper surface of the foliage. These lesions measure from one-fourth to one-half of an inch in diameter. Toward fall, in August and September, the lower surfaces of the leaves exhibit at first dark spots, which lack a red border, and soon develop finger-like cups in clusters. This is known as the cluster-cup stage of the rust fungus. Fruits are affected from the time when they are no larger than a pea (Fig. 94). The cups already mentioned for leaves develop anywhere on the fruit, although they are more common near the stalk end (Fig. 94). They are orange-colored and possess fringed margins. As a result of the affection, fruits are dwarfed (Fig. 94). These symptoms may also show on the apple and hawthorn, but less commonly than on the pear. Another stage of the fungus occurs on the red cedar. Galls are produced; they are an inch or less in diameter, very irregular surfaced, being marked

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by brown depressed scars. In the late spring dark-brown, wedge-shaped horns, which later become yellowish, are developed.

Cause.

The eastern pear rust is caused by the fungus *Gymnosporangium globosum.* Like other rust fungi it has two distinct host plants, the pear and the red cedar. If the spots of an affected pear leaf be examined, the upper surface will show small black fruiting bodies known as pycnia. These do not function in the life-history of the pathogene so far as is known, but are mentioned for their diagnostic value. They are found in the early summer. Shortly after their appearance another type of fruiting body develops on the lower surface opposite the pycnia. These structures are known as æcia. Occasionally they are found on the upper side of a leaf and even on the petioles. Ordinarily they are grouped irregularly over the affected area, although sometimes they are arranged in two rows, one on either side of the midrib. Æcia may also develop on the apple, hawthorn and mountain-ash. These bodies are deep cup-shaped, with their walls split part way to the base,

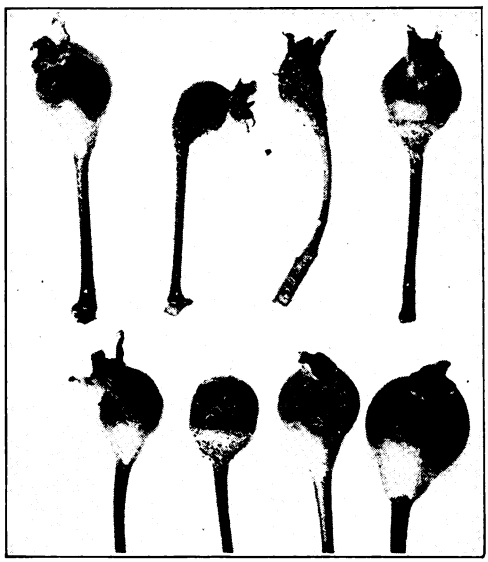

FIG. 94. — Eastern rust on pear-fruits.

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and are considerably larger and lighter colored than the pycnia. Within the æcia are developed æciospores which blow to and infect the red cedar. These spores may carry for a considerable distance. Cedar-apples are produced on the cedar as a result of æciospore infection. These cedar-apples are merely irregular, globoid galls measuring an inch or less in diameter. Their appearance has given rise to the popular term cedar-apples. The fungus mycelium developed from the æciospores grows within the tissues of the red cedar, stimulating them to over-growth, whence the gall. From the surface of the gall arise numerous short, beak-like horns which are composed of another kind of rust spores — the teliospores. In this rust fungus these spores may be produced year after year for several seasons from the same gall. When moist the beak-like horns become gelatinous and assume a bright orange color. In this gelatinous mass the teliospores grow out and produce a number of smaller spores called sporidia. This takes place in the spring. These sporidia are blown to the pear, apple or other host, where infection results.

Control.

Spraying for pear-rust is of little or doubtful value. Since the red cedar harbors the fungus, and since the fungus requires the presence of this plant in order to perpetuate itself, it is logical to destroy all red cedars within a reasonable distance. In the case of apple-rust the distance is put at one mile (see page 70), within which radius all cedar trees should be eradicated.

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Pacific Coast Rust

Caused by *Gymnosporangium Libocedri* (P. Henn.) Kern = *G. Blasdaleanum* (Diet. and Holw.) Kern

This is one of the recently recognized rusts of the pear family. The name Pacific Coast rust is used in contradistinction to eastern rust of pear (see page 341). The disease here considered was discovered in Oregon on cultivated pear several years ago, but was first given special attention in 1907. Since that date it has been found annually in Oregon and was particularly abundant in 1913. It is believed on good authority that this rust will in time become one of the most important pear troubles within its present range. It is most common on the native hosts, Oregon crab and its apple-hybrids, the hawthorn and service-berry. It also occurs on the ornamentals mountain-ash, Japanese quince, Japanese pear and flowering crab. It is common on the cultivated quince, but is rare on the cultivated apple.

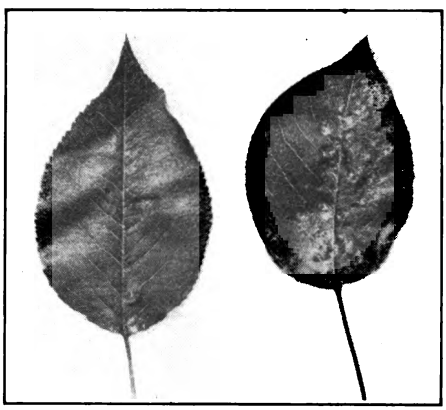

Fig. 95. — Pacific Coast rust on pear-leaves.

Symptoms.

The foliage (Fig. 95), fruit (Fig. 96) and stems are affected, and in each case there results some distortion. Yellow or orange spots develop on the affected parts; within the discolored area cup-like bodies with white margins are visible to the unaided eye. The fungus affects the incense cedar (*Libocedrus decurrens*), symptoms showing as brownish pustules on the leaves (Fig. 97). Some-

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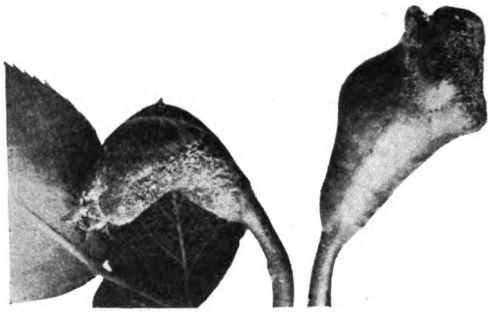

FIG. 96. — Pacific Coast rust on pear-fruit.

times witches'-brooms are formed; these may attain a diameter of two feet.

Cause.

The Pacific Coast rust fungus may properly be called *Gymnosporangium Libocedri*. Sporidia developed from teliospores on the incense cedar are blown for a considerable distance to the pear and other pomaceous fruits, where they cause infection. Within a few days after the pear is inoculated by sporidia, spots become visible on the affected organ as a result of infection. Within a month or less after inoculation mature æcia are present. From the æcia great numbers of æciospores are produced. These are liberated and are carried by the wind to the incense cedars, where infection results. The mycelium of

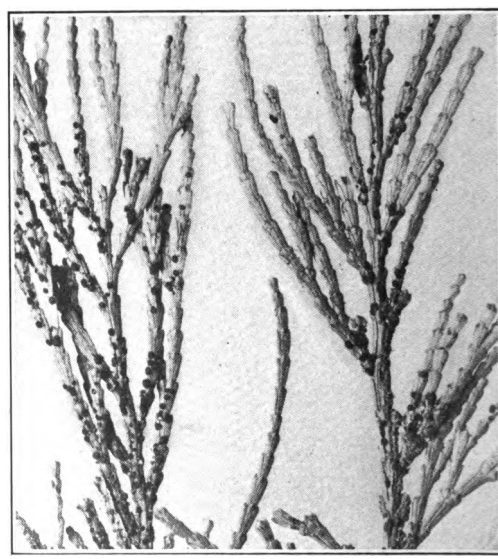

FIG. 97. — Pacific Coast rust of pear; telial stage on incense cedar.

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the fungus commonly stimulates the cedar to witches'-broom formation. Here in these tissues of the cedar the mycelium is perennial, the tissues retaining the fungus year after year without being infected anew by æciospores from a pomaceous host.

Control.

It is suggested that pear-scab sprayings (see page 335) probably would reduce this disease. Incense cedars should be removed where they are not highly valued, and pear-growers should avoid planting them. As a rule, however, this practice is unpopular, inasmuch as these trees are a valuable forest crop.

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Leaf-Blight

Caused by *Fabræa maculata* (Lév.) Atk.

This disease, which has been called scald and leaf-blight, is known in nearly all countries where the pear is grown. It is recorded from Germany, Sweden, Italy and France, and is especially common in eastern United States. In New York it is most abundant in the Hudson Valley and in the western part of the state, but perhaps even in these regions is less important than farther south and west. It is agreed that pear seedlings are most seriously affected; in some nurseries their cultivation has been greatly hindered, and in certain cases their growing has been abandoned. In Europe the leaf-blight has been known for at least a century, while in the United States it has

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been the subject of horticultural writings and discussions for many years. Very recently it was found on pears in Australia.

As already indicated, the disease is found more commonly in nurseries than in orchards, yet the latter are not exempt from attacks of the leaf-blight pathogene. In the nursery the trouble affects the leaves and twigs of seedlings; in the orchard, the fruits (Fig. 98), in addition, are susceptible. It appears that all varieties show the disease, yet the Kieffer and Angoulême are more resistant than the Seckel, Wilder Early and Sheldon. It has also been observed that the Flemish, Lawrence, Bosc and Clapp Favorite are resistant.

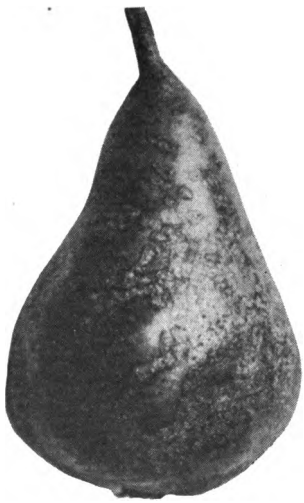

FIG. 98. — Leaf-blight lesions on pear-fruit.

Symptoms.

The disease makes its appearance early in the spring soon after the leaves develop. There develops a small circular, carmine-red spot, first on the upper, and then penetrating to the lower, surface. The color soon changes from red to dark-brown, with a slightly elevated, minute black spot in the center. If the lesions are numerous, they may merge, and thus the tissue between them turns brown. Affected young leaves shrivel; older ones change only in color. Badly diseased leaves turn yellow and fall prematurely.

The fruit shows the same carmine-red spot which afterward assumes a darker color (Fig. 98). The skin is roughened, and the growth of the epidermis is hindered, causing a deep crack in the flesh.

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The twigs, petioles and leaf-scales also exhibit signs of the disease similar to those on the foliage. The lesions on the twigs, however, are more elongated and become depressed, and finally girdling results.

Cause.

The leaf-blight disease is caused by a fungus, *Fabræa maculata.* Throughout the summer its conidia, developed upon the leaves, fruits and twigs, are scattered to other leaves, where infection results. It may be that certain of these conidia pass the winter on diseased twigs, producing infections the following spring. The chief method of hibernation, however, is by means of apothecial bodies in fallen leaves. Infected leaves, either at maturity or prematurity, fall to the ground in the late summer or autumn, carrying the fungus with them. Further activities on the part of the pathogene in these old leaves result in the formation of apothecia. In the spring, ascospores are discharged from these fruiting structures, and are carried to the susceptible parts. About one week later, the effects of the work of the parasite within the tissues are visible to the naked eye; and within a month, or less, after the ascospores are discharged, a new crop of spores, conidia, is developed. These conidia are borne in acervuli which are to be found in the center of each spot. Conidia from this source may infect the quince; likewise, the conidia from the quince may infect the pear. This is an adaptation on the part of the fungus which renders its control more difficult. Furthermore, these conidia may infect the hawthorn, apple and other closely related plants, thus adding complications to the application of remedial measures. All these plants, the quince, the apple and others, must be regarded as a source of trouble to the pears.

Control.

For the orchard trees and pear stocks, spraying is profitable and effective. Lime-sulfur 1 to 50 may be used with safety and with success. It is recommended that iron-sulfate be

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added to the above, at the rate of 3 pounds to 50 gallons, to increase the adhesiveness of the fungicide. The first application should be made soon after the first leaves develop. There should be four or five applications subsequent to the first, depending upon the season. In seasons with only moderate rainfall an application should be made once in every three weeks.

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(See additional references under Quince, page 390.)

OZONIUM ROOT-ROT

Caused by *Ozonium omnivorum* Shear

This root disease is confined to the South and Southwest, having been found particularly in Texas and New Mexico. Pears are frequently injured to a very appreciable extent. Affected trees usually show, on the roots, threads of the causal fungus, *Ozonium omnivorum;* these threads are at first whitish, then dirty-white or brown. Pear-trees in close proximity to affected cotton, which also is affected by this disease, are most liable to infection. It is believed that a tree once diseased cannot be saved. (See under Apple, page 146.)

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Sooty-Blotch and Fly-Speck

Caused by *Leptothyrium pomi* (Mont. and Fr.) Sacc.

The pear fruit is sometimes affected by the sooty disease which occurs so commonly on the apple in a wet season. It is rarely troublesome to the pear-grower. (See Apple, page 104.)

Black-Mold

The fungus *Fumago vagans* Fries

The leaves, twigs and fruits of pears, apples and cherries are sometimes badly discolored by the growth of the fungus *Fumago vagans* in the honey dew secreted by lice. The fungus is dark and its development is so extensive that it presents a very conspicuous black or sooty mold aspect. Since *F. vagans* lives in the honey dew, it is most prevalent in epidemics of the pear psylla and apple louse. The fungus itself needs to cause no fear; it may be eliminated by controlling the above-named insects.

Black-Rot Canker

Caused by *Physalospora Cydoniæ* Arnaud

The fruit, leaves and branches of the pear are attacked occasionally by the above-named fungus, and as a result a black-rot, a leaf-spot or a canker may be produced. Special reports of it have come from New York, Texas and Ohio, and it doubtless has a wide range over the eastern United States. The causal fungus, *Physalospora Cydoniæ*, is very common, infesting dead bark of many woody plants. It readily attacks healthy bark of the pear and apple whenever an injury affords entrance. In this connection it has been suspected as the cause of the failure of grafts, killing the end of the stock in

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which the scions are set. This type of trouble, while itself not common, illustrates what a wound parasite like *P. Cydoniæ* is capable of doing. (For fuller discussion see Apple, page 45.)

Brown-Rot

Caused by *Sclerotinia cinerea* (Bon.) Schröt.

Outbreaks of brown-rot due to the above-named fungus are not at all common in America, although European growers encounter a similar trouble frequently. It is better known on the apple than on the pear in this country. The blossoms, fruits and branches are susceptible, and when affected the various phases are referred to respectively as blossom-blight, brown-rot, and brown-rot canker. Recently a brown-rot canker has been described from Oregon, but it has not been definitely determined whether the fungus concerned is *S. cinerea* or another species.

Bitter-Rot

Caused by *Glomerella cingulata* (Stoneman) Sp. and von S.

Fortunately for the pear-grower bitter-rot is never as common and destructive to the pear as to the apple. Whereas apple bitter-rot ranks second in importance only to apple-scab over the country as a whole, pear bitter-rot is rare and has never been found doing extensive damage. The fungus, *G. cingulata*, was reported as causing cankers on both young and old trees in Texas a few years ago. Characteristics of the disease on the fruit and woody portions of the tree, together with a description of the life-history and control of the pathogene, are given in some detail under Apple, page 14.

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Red Leaf

Cause not definitely known

This peculiar and interesting disease has been observed in New York State during the past five or six years. There are no extensive data as to its preference for varieties, although it has been observed especially on the Kieffer and Clairgeau. In some cases under attention the orchards showing red-leaf, or crimson foliage, were located on a hillside, and the disease was confined to those individuals occupying the lower part of the orchard. Such trees also exhibit signs of general unthriftiness. Affected trees stand out prominently among their healthy neighbors and exhibit an entire foliage discoloration; a dark or purplish red color. The suggestion has been made that lack of vigor, of which red-leaf is a sign, may be due to one or more factors. Such factors worthy of note are poor underdrainage, insufficient plant food, a diseased condition of the trunk or roots, and winter and drought injuries.

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CHAPTER XII

PLUM DISEASES

THE plum furnishes food and habitation for several parasitic fungi and bacteria. It is also affected with other ills caused by frost and other factors not generally understood. Many of these diseases are native in their origin, so that often European plums are highly susceptible to important diseases in America.

BROWN-ROT

Caused by *Sclerotinia cinerea* (Bon.) Schröt.

No disease gives the commercial plum-grower so much trouble as brown-rot. It occurs more or less every year, affects the ripe or ripening fruit, and destroys it absolutely (Fig. 99). It is equally annoying and destructive in small home gardens, rotting the fruit just at the time when it is ready for use.

It is said that thin-skinned varieties are more resistant to brown-rot than others. It is also said that the Triflora, Americana and Japanese groups are more injured than plums in other groups. The Burbank, Abundance and Satsuma are said to be most susceptible, while several varieties are listed as resistant. Among the latter may be noted: Hansom, Clinton, Forest Rose, Indiana, Miner, Nebraska, Prairie Flower and others. It is to be noted that most of the list of so-called resistant plums are varieties of minor importance; at least they are not leading American plums. And it is doubtful whether the Bur-

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bank, Satsuma and others suffer a great deal more than other prominent varieties. Careful observations along this line seem desirable.

Brown-rot of plum is a disease also found on the peach and cherry (see pages 270 and 170, respectively). On these last two fruits the disease is perhaps more destructive than on plums. The common occurrence of the disease on all these hosts gives it a wide range over this and other countries. Losses in various parts of the United States are estimated at 30 to 100 per cent on susceptible plums. At times twig-blight is caused by the fungus (*Sclerotinia cinerea*); this type of injury has been reported from Iowa and New York. The Red June, Wickson and other upright-growing varieties are peculiarly susceptible to the twig-blight form of this disease. In the Pacific Northwest blossom-blight and mold in transit are important phases of brown-rot on prunes.

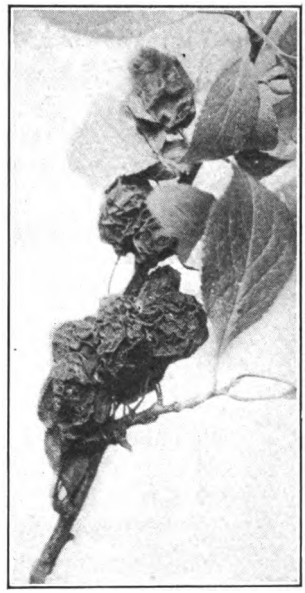

FIG. 99. — Group of plums mummified by the brown-rot fungus.

A more complete discussion is given under Peach, page 270.

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(See additional references to literature on brown-rot under Peach, page 276, and under Cherry, page 172.)

Black-Knot

Caused by *Plowrightia morbosa* (Schw.) Sacc.

The black-knot, or plum-wart, is a conspicuous disease of the plum and cherry, affecting both the wild and cultivated forms. It appears that, on the whole, plums suffer more than cherries. It sometimes happens that when wild plums and choke-cherries are growing in close proximity, even with their branches intermingled, the one may be affected with black-knot, whereas the other shows no signs of the disease. This is explained on the grounds that the pathogene has become so adapted to growth on the plum, for example, that it is not capable of attacking and infecting the cherry, and vice versa. There has always been more or less discussion of the resistance and susceptibility of various varieties, but on the whole varieties within a species do not show marked differences in this respect. The Trifloras are said to be affected less than any other group of plums, and the Institias rank next in immunity, although the Damson is said to be very susceptible. On the other hand, the Domesticas are susceptible, except possibly the Middleburg and Palatine, which are relatively free from black-knot. Further observations of the whole question of varietal susceptibility are desirable.

Black-knot is of native origin and has been the subject of horticultural and botanical writings since the beginning of the

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nineteenth century. It is not possible to state accurately just where the disease originated, but the first records show that many years ago black-knot was particularly abundant along the northern half of the Atlantic seaboard. The evidence at hand indicates that the disease may have first affected cultivated forms of the plum and cherry in Massachusetts about 1800. It may be pointed out, however, that this portion of the East was first most thickly settled and consequently it was there first noticed. The disease was confined to the eastern United States until about 1879, when the pathogene spread westward, appearing in the vicinity of Cincinnati, Ohio. This invasion of the western states has continued uninterruptedly, and now the disease is found across the northern United States to the Pacific Coast. It occurs less commonly in large portions of the Southwest and Central West.

The disease is one of the most common of stone-fruit tree troubles in America. It is as destructive as it is common. It is of great economic importance because of its wide geographical range and on account of its prevalence for the past century. The loss in dollars incurred during this long period would be difficult to determine. But it is interesting to note that the destructive nature of black-knot caused plums to be so scarce in New England in the autumn of 1875 that $2.50 was paid for a peck of Damsons in the city of Boston. At one time the raising of cherries was almost abandoned in the State of Maine on account of black-knot. The disease has been equally destructive in New York. In one plum orchard which, in 1884, netted $8000, thousands of trees were rooted out in 1885, and from the remaining ones the sum of but $250 was realized. Numerous similar cases might be cited, not only for New York, but for many other states within the geographical range of the disease. Destruction in any degree is a direct result of the death of affected twigs, limbs, and occasionally the trunk.

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Symptoms.

The disease affects only the woody parts of the host, and usually only the twigs, although the pathogene may spread from affected spurs into the larger limbs or the body of the tree. The normal form of the diseased part is strikingly changed (Figs. 100, 101, 102, 103 and 104). The knot is usually fusiform, but sometimes it may be the same diameter throughout its length, in which case the knot terminates abruptly. As the name suggests, black knots are produced. A knot begins at any of the following places: (1) near the tips of twigs (Fig. 100); (2) in the crotches of younger limbs at the union of the consecutive growths of two seasons; (3) on small spurs which commonly bear the fruits (Fig. 100); (4) near the axil of a leaf; and (5) in the crotches of limbs four or five years old. Knots vary from one-half of an inch to a foot or more in length and from a fraction of an inch to two inches in circumference. Usually the knots do not extend around the limb (Fig. 103), although in some cases they completely surround the affected part (Fig. 104). When their course is long, they tend to proceed spirally about the stem (Fig. 101). In the spring young knots are olive-green in color and at this time are solid but rather pulpy. As the season advances the knots become harder, more brittle, and their surfaces become black. Frequently older knots are attacked by insects which destroy the central part in the knot, leav-

FIG. 100. — Small black-knot swellings about buds.

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ing the black outer shell. On plums the interior of an old knot is honey-combed. Final stages in the development of the knot show the exudation of gum and the growth on the surface of a pink mold.

An interesting response is shown in the case of twigs affected near their tips. When the knot so develops, the twig becomes bent so that a right angle will be made from the knotted side. Sometimes, in the case of affected branches which are not killed, a swelling is produced just above and below the knot.

Cause.

The black-knot disease is caused by the fungus *Plowrightia morbosa*. Its action stimulates the tissues of the twigs and limbs to form the characteristic galls or knots. The fungus begins its work by disseminating its spores in the spring. This process is promiscuous, but some of the spores find lodgment upon the plum or cherry. At various points, already enumerated, the knots have their beginning. With the growth of the spore there develops a system of vegetative threads which pervade the bark and which very soon attack the growing tissue (the cambium) between the bark and the wood. The fungus irritates this region and the tree responds by forming not only an excessive amount, but also an irregular arrangement of the bark tissues (Figs. 103 and 104). This irregularity of development in the affected region pro-

FIG. 101. — Black-knot; one-year-old knot with a brownish velvety surface.

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ceeds to such an extent that the normal form of the limb is strikingly changed (Fig. 102). The newly forming knot can often be detected in the fall, when it appears as a slight swelling (Fig. 100). It is, however, more conspicuous in the spring; at this time it enlarges and the bark is ruptured, thus exposing a yellowish surface (Fig. 101). This color does not prevail long, but the fungus grows to the exterior and thereon develops its summer spores which give to the knot a velvety olivaceous appearance. These spores are disseminated in April, May and June, being carried by the wind to suitable places for initiating a new knot. As the season advances the gall gradually changes color; by the first of September black dots appear over the surface, and within another month the whole knot is perfectly black and presents the appearance which is so commonly seen (Fig. 102). If one examines such a knot closely, it will be observed that it is then covered by innumerable small elevations which project from its irregular surface (Fig. 102). Each of these elevations constitutes a winter condition of the fungus — a perithecium in which a second kind of spores develops from

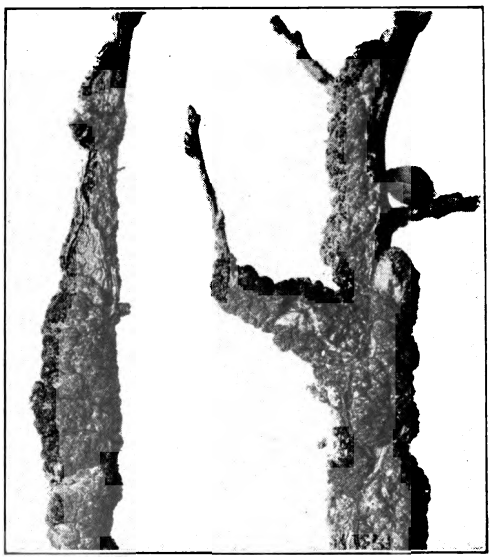

FIG. 102. — Black-knot; two-year-old knots with black roughened surface.

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January to June, depending upon the locality, and which are discharged upon maturity. The further history of these spores is similar to that of the summer spores previously described; they propagate the parasite. The original portion of the knot is thus matured, but the fungus in the bark may continue to grow at either end, thereby increasing the length of the old knot. In this manner a single knot may in time extend a long distance on a branch, its course tending to proceed spirally about the stem.

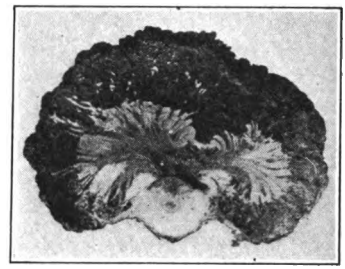

FIG. 103. — Black-knot; cross-section of a knotted twig. The enlargement has not completely encircled the twig.

Control.

Eradication of the causal fungus is the recognized method of most practical value in the control of black-knot. In following this principle, it should be remembered that (1) the summer spores are produced abundantly during the late spring and early summer, (2) the winter spores develop from midwinter to spring, and (3) there are many kinds of plums and cherries attacked by these spores. Therefore, prune out the knots before either kind of spores is matured, that is, in the fall or early winter, before January. Look for them in the wild as well as in the cultivated species of plums and cherries. An annual fall inspection of the trees and the removal and destruction of all the knots is the most

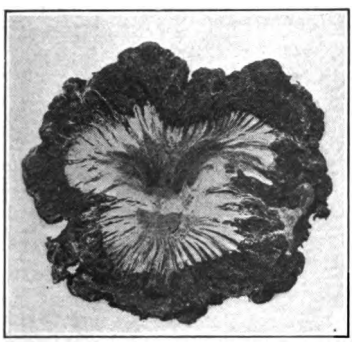

FIG. 104. — Black-knot; cross-section of a diseased twig. The knot has completely encircled the twig.

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satisfactory program of eradication. There is no objection to more frequent inspections, but a single annual inspection, if carefully done, should prove sufficient. It is necessary to destroy these knots, otherwise the spores contained therein are likely to be liberated and start the trouble again. This method of control has not given the satisfaction that it should, the difficulty apparently lying in the carelessness of the inspector and the failure of the neighbor to cooperate. It is obvious that if but a few knots are overlooked, the fungus may still operate successfully. And the growers of a given region must make the plan one of joint-action; one individual cannot exterminate the fungus in a locality. If the existing laws were enforced, the extent of injury from this disease would be greatly diminished. The reward for any such eradication measures will be reaped in accordance with the vigilance of the local orchardists.

As a measure purely supplemental to the one just outlined the trees should be sprayed with bordeaux mixture, the number of applications depending upon the severity of the case in hand. In cases of a threatening nature the sprayings should begin late in March; five or six subsequent applications at intervals of two or three weeks should be made.

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Leaf-Blight

Caused by *Coccomyces Prunophoræ* Higgins

Leaf-blight, leaf-spot, shot-hole and yellow-leaf are all names referring to the disease under consideration. A very similar disease affects the cherry (see page 172).

Among plums the European varieties are notably susceptible to leaf-blight; these may be completely defoliated in wet seasons. On the other hand, Japanese plums and Myrobalan seedlings are comparatively resistant, the disease being of little consequence thereon.

Affected leaves show dead spots at first (Fig. 105); but soon the dead tissue falls away, leaving a circular hole, whence the name shot-hole (Fig. 105). This type of symptom is common on the plum. At times leaves are completely riddled with holes. Sometimes affected leaves turn yellow, whence the name yellow-leaf. The similar disease on cherry shows less shot-holing and more yellowing of the leaves than in the case of plum. In most seasons if sufficient moisture is present, white, velvety pustules appear on the lower surface. These are coils of spores belonging to the conidial form of the causal fungus *Coccomyces Prunophoræ* (commonly known as *Cylindrosporium Padi*).

Bordeaux mixture 5–5–50 is effective in the control of plum leaf-blight, but this fungicide may injure the foliage, particularly in nursery-stock. Lime-sulfur, on the contrary, diluted 1–50, may be used without fear of such injury. In the orchard, spray as follows: (1) ten days after the blossoms fall; (2) three weeks after the first application; (3) three to four weeks after the

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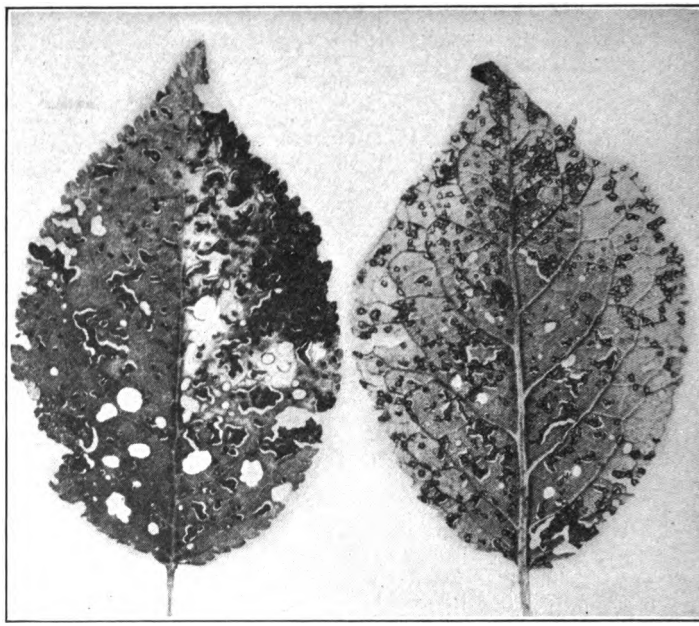

FIG. 105. — Leaf-blight of plum. Note the shot-hole effect. Upper surface (left), lower surface (right).

second application. In relatively dry seasons the third spraying may be omitted. Treatment for nursery trees should be given as recommended under Cherry, page 175.

See fuller discussion of the symptoms and cause under Cherry, page 172. The leaf-blight of plum and cherry, while not caused by identical fungi, are so similar that the statements made for cherry leaf-blight apply to the plum.

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See further references to literature under Cherry, page 176.

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Frost-Injury

Caused by the action of low temperatures

Japanese plums behave much as do peaches when injured by frost. Domestica plums, on the other hand, are more hardy, except the variety Reine Claude, which is highly susceptible to sun-scald. (See Apple for fuller discussion, page 35.)

Die-back

Caused by *Valsa leucostoma* Fr. var. *rubescens* Rolfs

This very common disease of stone-fruits, in particular, affects the plum, resulting in a dying back of the twigs and often in the formation of large cankers on larger limbs. See more detailed discussion under Peach, page 300.

Gummosis

Various causal factors

Like other stone-fruits, the plum is subject to gum-flow when injured in any way. (See in this connection the discussion presented under Peach, page 303.)

In the Pacific Northwest a bacterial-gummosis has recently been studied, and the cause found to be *Bacterium Cerasi* (= *Pseudomonas Cerasus* Griffin). Particular attention, however, was given to it as it occurred on the cherry. The disease on the plum is similar to the bacterial-gummosis of the cherry. This trouble on the plum is often erroneously called winter-injury and sun-scald, but it has been shown that in most cases at least the action of the sun has nothing to do with the production of this disease. Cankers similar to those on the cherry are produced. Such lesions on the plum, however, exude noticeably less gum than those on the cherry.

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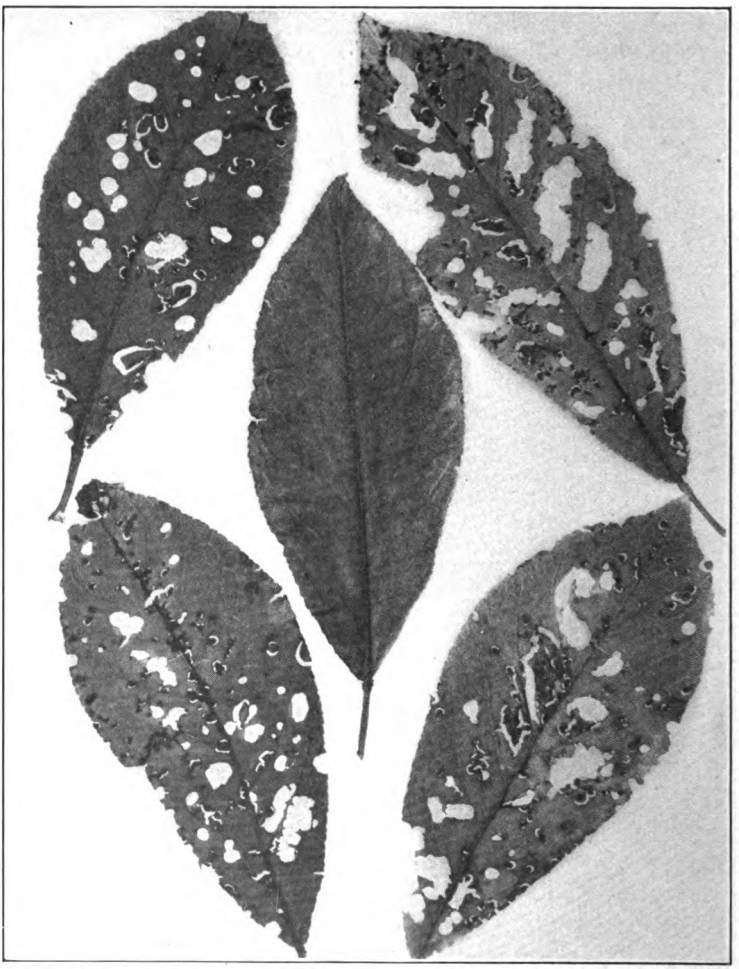

FIG. 106. — Black-spot lesions on plum leaves; healthy leaf in center.

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BLACK-SPOT

Caused by *Bacterium Pruni* E. F. Smith

This is a disease which occurs on the peach, apricot and nectarine as well as the plum, and which is known as black-spot, shot-hole, bacterial leaf-spot and bacterial-crack. Of all fruits affected, plums show the greatest variation in susceptibility of varieties. The larger part of American plums suffer but little, whereas Japanese varieties, especially Abundance and Burbank, are severely injured. The Chabot and Red June are among other susceptible varieties. The more resistant plums include Arkansas, Clifford, Cumberland, Diamond, Damson, Lombard, Wild Goose and Yellow Egg.

Black-spot of plums has been noted in various sections of eastern United States during the past ten or twelve years. It was first observed in Georgia in 1904, in Nebraska and Missouri in 1906, in Delaware in 1907, and on Long Island about 1908. Subsequent records have been made east of the Mississippi River.

The leaves (Fig. 106), fruits (Fig. 107) and twigs are diseased. The leaves show a spot in which the tissue is brown and dead (Fig. 106). Soon the affected portion falls away, leaving a shot-hole. The holes are comparatively large and irregular in outline (Fig. 106). In severe cases premature defoliation occurs. Fruits of susceptible varieties are badly distorted and cracked, and are thus rendered liable to rot-producing organisms. Black spots are developed thereon (Fig. 107). On plum-twigs it is characteristic for the black spots to persist from year to year,

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forming perennial cankers. Thus old neglected trees are particularly a constant source of trouble and danger to other trees. These cankers are relatively large and deep on the Abundance and Burbank varieties. Cankers on the plum are somewhat different in appearance from those on the apricot, nectarine and peach. Open cankers are the more common type observed on the last three fruits, whereas on the plum the affected bark clings to the twig for some time. Watersprouts of plums may at times show twenty or more cankers in various stages of development.

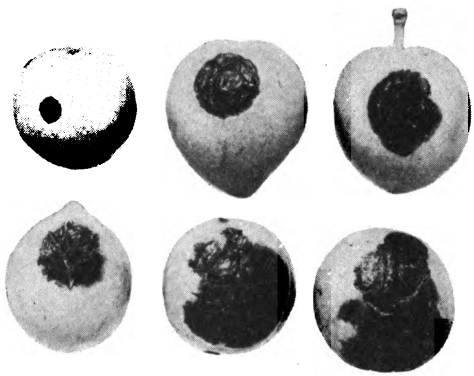

FIG. 107. — Black-spot on plums; developmental stages.

See more complete account of Black-Spot under Peach, page 306.

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(Additional references to literature under Peach, page 311.)

SILVER-LEAF

Caused by *Stereum purpureum* Fries

While this disease has not been observed with certainty on plums in the United States, it is known on apple, and its prevalence and destructive nature in Canada and elsewhere would

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seem to warrant an account here. The disease is known in Canada from Nova Scotia to Vancouver Island, and in Germany, France, England, South Africa and New Zealand. It has been the subject of observation and investigation since 1885, when it was described in France. Outside of the United States, the disease is said to be one of the most widely distributed of plant diseases. While silver-leaf occurs chiefly on the apple in Canada, it is primarily a plum trouble in other regions where it prevails. The disease, known also as silver-blight and silver-disease, affects, in addition to the plum and apple, many other fruit-trees and bushes, as well as certain nut and forest trees. Among these may be noted: peach, pear, cherry (wild and cultivated), currant, gooseberry, almond, chestnut and ash.

Of all the fruit-trees the plum is said to suffer most. Among the plums it is quite probable that all varieties are equally susceptible to the spread of the pathogene, once the tree is infected. It is a common notion, however, that soft-wooded varieties are more commonly affected. In many instances this may be explained on the grounds that such varieties are in the majority in a given orchard or locality. On the other hand, if soft-wooded varieties are more susceptible, the explanation may lie in the fact that they are more susceptible to injuries, through which the attack is made. Reports have it that the Victoria, Gibson, Wales, Oullins, Early Rivers, Flemish, Czar, Monarch, Orleans, Washington and Damson suffer more than other varieties. Those said to be free from the disease in orchards where silver-leaf occurs are: Diamond, Jefferson, Reine Claude, Denniston, Mirabelle, White Bullace, Sultan, Wales, Englebert and Early Rivers. It will be perceived that certain varieties have been observed by different individuals to be in one case susceptible, and in another, resistant. This is not surprising, since so many factors must be taken into account in the consideration of susceptibility and resistance of varieties to any given disease.

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Symptoms.

In the beginning the disease is confined to a single branch or twig; but from year to year other branches are affected until the whole tree is involved. Very frequently one or more twigs are killed before the trouble extends to the rest of the tree. In many cases three to six years elapse before the tree is wholly killed.

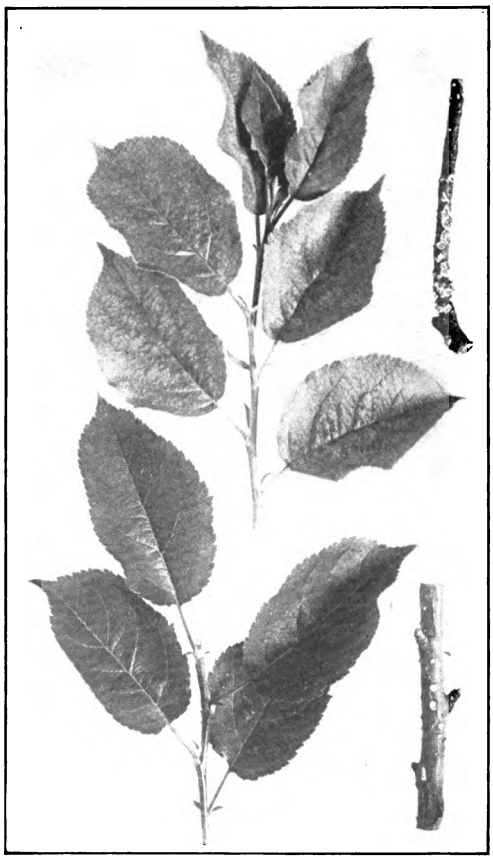

FIG. 108. — Silver-leaf: leaves on upper shoot silvered; leaves on lower shoot healthy. Twigs at right showing fruiting bodies of the causal fungus.

The external signs of the disease are confined to the leaves. Affected foliage generally remains normal as to size and form, but the surfaces, instead of exhibiting the normal green color, show a peculiar ashen gray luster; the color more nearly approaches that of lead than any other which has been suggested (Fig. 108). This peculiarity is noticeable at a great distance from the trees as well as upon closer exami-

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nation. After affected trees have died, fruiting bodies of the causal pathogene appear (Fig. 108). The limbs and roots are affected, but give no external evidences of the disease. But on cutting into these parts the wood will be found to exhibit a brownish discoloration. Frequently discolored hearts are not accompanied by silvery foliage, and vice versa. In certain cases the discoloration of the wood is due to some other cause than the silver-leaf pathogene. As a general rule only about 33 per cent of the affected trees recover from the effects of the disease, although cases are on record where a tree once entirely silvered finally recovered.

Cause.

Although the silver-leaf disease has been known for many years, the true cause was not definitely determined until 1902, when the fungus *Stereum purpureum* was discovered in this rôle. Some had held that bacteria were the causal factor involved, while others dismissed the question with the statement that the silvering of plum leaves was the result of some disturbance in the nutrition. Other more or less fantastic speculations regarding the cause of the disease are encountered in a review of the notions held on the subject.

Trees become inoculated above and below the ground; that is, the fungus may enter the tree through the branches or trunks, or through the roots. In general the trunks are the more liable to attack. Spores are blown to the aërial woody parts, where they germinate and penetrate through a wound of some sort. The fungus is not carried by pruning tools; it is not disseminated by means of buds or scions from a diseased tree. However, it should be noted in connection with the latter point that scions from healthy trees when grafted on diseased stock become diseased, as evidenced by their silvered leaves. The spores, which are carried to the trunks and branches, may come from any of the long list of plants which happen to be dead as a result of the attacks of *Stereum purpureum*. The fruit-bodies (Fig.

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108) develop only on dead trees, either standing or in the brush-heap. They also occur on stumps of trees once silvered. So far as root-infection is concerned, the mycelial strands are easily carried in cultivation; they are torn from the roots of affected trees, transferred to roots of neighboring trees, where entrance is gained through an injury made by the cultivator. As intimated above, leaves are never directly affected. Trees infected in the fall show signs of the disease in the spring of the following year. Those infected earlier in the spring or summer may show silvering within one or two weeks, or in some cases only after two months. In all cases of branch-infection the leaves above the infection-court show more silvering than those below the point of attack. And only those leaves on the same side as the infection-court show the disease, those off a straight line from the point of inoculation to the tip remaining normal. It appears then that the real disturbing factor concerned in the silvering of the foliage is conducted rapidly in the sap. This is further supported by the fact that the mycelium of the fungus has never been observed within the leaf-tissues. The hyphæ are found, however, in the roots, trunks and branches, in the xylem vessels. Here the walls of the vessels and of the medullary ray-cells are brown and their lumina are filled with a brownish red substance. The cause of the silvering in the leaves is thought to be due to some poisonous substance secreted by the mycelium in the woody portions. There is no alteration of the chloroplastids in the leaf-cells; the normal structure of the leaf is not markedly changed. The epidermis is enlarged, and raised, the elevation resulting in the formation of air spaces between the epidermal and mesophyl layers. Thus the chlorophyl cannot show through the surface; instead, the air spaces give to the leaf surface a lead-colored or silvery appearance.

As previously noted, most trees die, after which the fungus develops its fruit-bodies. These are long-lived, being able to withstand at least 13 months of dry weather. On moistening again, mature spores are ejected.

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Control.

The customary measures of fruit disease control do not readily apply to silver-leaf. As a matter of fact, no specific schedule for the control of silver-leaf, based upon experimentation, is at hand. Certain precautionary measures should be taken: (1) affected limbs, trunks and stumps should be wholly destroyed, and brush-piles should never be allowed to accumulate; (2) avoid the use of the limbs or trunks of any of the previously-mentioned fruit-trees as props and posts; (3) avoid injuries to the roots, and to the trunks and branches; (4) avoid planting young trees showing brown hearts.

It has been shown that the application of iron sulfate, as used in New Zealand, is wholly without value in the control of silver-leaf.

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POCKETS, OR BLADDERS

Caused by *Exoascus Pruni* Fckl.

This disease has a very general geographical range in Europe and in portions of the United States. It has been reported from Michigan, Iowa, South Dakota, North Dakota, Ohio,

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Colorado, New Jersey, North Carolina, New York, Kentucky, Kansas and other states. The disease was observed as early as 1593, but it attracted little attention in America before 1888, at which time it occurred commonly in certain of the states already listed. The history of the disease indicates that it may never be expected to cause serious trouble over wide areas. It has never swept over any country, but rather affects individual trees here and there in isolated localities. In many cases, however, affected trees may not have a single healthy plum. Moreover, once a tree is diseased it seldom wholly recovers, but is attacked from year to year. The nature of the losses is as follows: (1) plums are distorted (Fig. 109) and may be rendered unmarketable; (2) affected fruits drop prematurely; (3) leaves are curled; (4) twigs are swollen, distorted and finally killed.

All kinds of plums are attacked, although the trouble is more abundant on the red and purple varieties. Wild species are said to be more commonly affected than cultivated ones. It is to be noted in connection with susceptibility that there is an apparent individual resistance which may be confusing in pointing to resistant and susceptible varieties. Certain individuals of a given variety may be seriously affected, whereas others of the same variety, in close proximity to the affected trees, remain unaffected.

Symptoms.

In the case of affected fruits, there results, instead of normal plums, peculiarly enlarged, hollow deformities (Fig. 109). It is this condition which has given rise to the name plum-pockets. These consist merely of a thin shell, and in most cases there is no evidence of a seed. Wherever a stone is present at all, it is only rudimentary. The disease makes its appearance soon after the flowers have fallen. Toward the last of June the disease attains its full course of development and affected fruits drop from the tree. Affected plums are at first globoid in shape, but as they grow older they become oblong or oval, and more or

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less curved (Fig. 109). When young they are smooth, and can be distinguished from the healthy fruit by their pale-yellow or reddish color. Later, the surface is gray and is wrinkled considerably. Finally, diseased fruits turn dark-brown or black, and rattle like bladders when brought in contact with any hard substance; whence the name plum-bladders. In this condition they hang to the tree two or three days, then fall to the ground.

Fig. 109. — Plum-pockets.

The younger branches and leaves are affected, which increases the damage done to the tree. The growing limbs and leaflets become distorted and swollen. In June the diseased branches turn gray, the tissues soften, dry up and die. Shoots arising the next year just below these dead extremities are most frequently affected by the disease. As the new leaves unfold they turn red or yellow, when affected by the disease, and show an arching of the leaf-blade. In general a curl disease, similar to peach leaf-curl, is developed.

Cause.

The cause of these interesting malformations is the fungus *Exoascus Pruni*. Its growth in the plum-tissues causes their peculiar development, which finally result in the so-called pockets. For a long time the cause of the disease was not known, but was attributed to the work of some insect, to improper fertilization,

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and to a superabundance of moisture in the atmosphere at the time the fruit was forming. The real cause of plum-pockets was discovered in 1861.

The parasite spends the winter as mycelium in the smaller branches, as evidenced by the fact that it is found there before diseased fruit appears. Moreover, the annual recurrence of the disease strengthens this opinion. From the affected branches the mycelium grows out into the young ovaries of the fruit, stimulating them to form a remarkable hypertrophy. After the mycelium develops to a considerable degree internally, the threads pass towards the surface. Some of the hyphæ push up between the epidermal cells and spread out between these and the cuticle. Here they form a net-work of short cells, which soon grow erect on the surface of the fruit and become asci with ascospores. The ascospores are discharged through the apex of the ascus, but their further history is unknown. Conditions favorable to plum-pockets are similar to those favoring peach leaf-curl, namely cool, wet weather in the early season of the year.

Control.

Satisfactory control measures have not been established for all sections of the country. It is advised that severe pruning to remove diseased twigs be practiced. It has been found in Montana that trees sprayed with lime-sulfur showed very few diseased plums, whereas more than half of the fruit on unsprayed trees was destroyed. These results were obtained in 1915 by making two applications as follows: (1) just before the flower-buds open, and (2) just after the petals fall. This treatment is advised for other regions where the disease is troublesome.

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Rust

Caused by *Puccinia Pruni-spinosæ* Pers.

On the leaves of many stone-fruits is produced a rust disease known as plum leaf-rust, prune-rust and rust of stone-fruit trees. The disease is widely distributed in North America, Europe and Asia, and is common in South America, Africa and Australia. In the United States it is reported from most of the states east of the Mississippi River, and from Iowa, Missouri, Oklahoma, Texas, Louisiana, California and Oregon. It doubtless occurs in other states of the Union. In Europe it is found in Germany, France, England, Italy, Austria, Belgium and Switzerland. These statements indicate the cosmopolitan occurrence of plum leaf-rust throughout the world.

Outbreaks of this disease are not uncommon. In 1886 the troublesome nature of plum-rust attracted attention in Australia; in 1889 it was serious in Iowa; and in recent years it has been the object of experimentation in the far West. In severe cases defoliation may occur; this is true of the prune and peach in California and of the peach in Texas. It is more destructive in warm, moist climates where crops are seriously impaired and where trees are said to die within three or four years after the attack. In the United States most damage is done in the southern portion, and of all trees affected the plum suffers most. Chippewa plums are said to be particularly susceptible. Likewise the Imperial Ottoman is badly affected.

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Symptoms.

The disease appears in midsummer but is most abundant in fall. Only the leaves are commonly affected; fruits or other parts of the plum rarely show rust-lesions. On the lower surfaces rust-pustules are found (Fig. 110); these are light-brown, small, round and somewhat powdery. They are scattered, or they may be so numerous as to practically cover the leaf (Fig. 110). The development of these pustules is preceded by the formation of yellowish spots. Later the pustules, or sori, to be observed on the leaves are dark-brown or almost black, but they still retain their powdery nature.

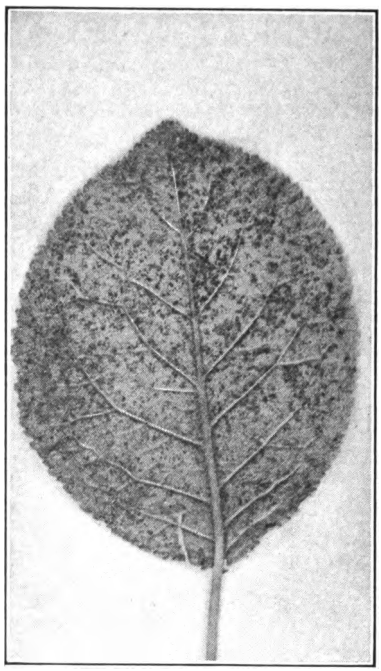

FIG. 110. — Plum-rust, on lower surface of leaf.

Cause.

The pustules or sori just described are fruit-bodies of the causal fungus, *Puccinia Pruni-spinosæ*. Within the affected leaves of the plum the mycelium of this fungus is found. It comes to the surface and there forms sori, within which are developed first a crop of spores known as uredospores. These are capable of infecting other plum leaves as well as the foliage, and in some cases the fruit of the peach, almond, cherry, apricot and nectarine. On all these hosts, too, uredospores are developed which are capable of infecting the plum. These spores may

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winter-over, are viable in the spring, and can cause new infections after several months' rest.

After the uredospores have been developed they are replaced in the sori by other spores, known as teliospores. These follow closely the development of uredospores; they are said to appear in five to twenty days after the uredospores. The teliospores germinate in the spring, producing small, light sporidia which do not infect the stone-fruits, but must go to species of Anemone, Thalictrum and Hepatica, all common wild flowers. On any of these flowers germination and infection occur. The mycelium developed in these hosts lives over until spring, when fruiting sori-bearing æciospores are produced. Just preceding these bodies sterile structures known as pycnia are developed on the upper surface of the leaves as black dots. The mycelium in the leaves may live over in these wild flowering plants for several years in succession, so that the stone-fruits are not absolutely necessary to the perpetuation of the fungus on these wild flowers. In this connection, however, it should be noted that the wild flowers already enumerated are necessary to the perpetuation of the fungus on stone-fruits. The æciospores developed on Anemone and others are wind-borne to the plum, where infection occurs on the leaves. Within three weeks after inoculation uredospores are mature and ready for summer dissemination of the fungus. Plum-trees are not generally attacked until sometime after the month of June, except in Australia, where infection occurs in February and March.

Control.

In those sections where plum-rust is troublesome it is probable that spraying will reduce the injury. The treatment as advised for peach leaf-curl, that is, spraying before the buds open with some standard fungicide, is recommended. In southern California it appears that early fall pruning performed to such an extent that fall growth is stimulated is not advisable. The difficulty involved lies in the fact that such foliage remains alive

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through the winter, and rust developed thereon permits early spring infections which bring about considerable damage to the orchard. In Oregon it is thought that bordeaux spraying the last of August or the first of September will prove profitable.

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WILT

Caused by *Lasiodiplodia Triflorœ* Higgins

Apparently this disease is very limited in its geographical range. Reports of it come from only three states, Georgia, North Carolina and Alabama. In this region, however, it is of considerable importance, and in Georgia more especially it is regarded as an important factor in commercial plum-growing. Japanese plums and their hybrids are particularly sensitive to wilt. The disease appears to have been known for several years.

Symptoms.

Plum-wilt (Fig. 111) resembles fire-blight and for some years was thought to be of a similar nature. Leaves on a branch or a whole tree suddenly wilt in the spring or early summer. Examination of the base of the wilted portion will reveal dark, dead bark. Frequently this dead area extends along one side of the trunk to the surface of the ground. Within a year from first wilting, the whole tree may die.

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Before wilting, diseased trees may be located by the scorched appearance of their leaves. This is particularly noticeable in dry weather in late summer. The edges of the leaves become dry. This, however, is not a specific sign of the wilt disease inasmuch as this appearance may be induced by any factor which cuts off the water-supply. Trees growing in a dry, hard soil as well as those with diseased roots are very liable to exhibit symptoms similar to those of the early stages of wilt.

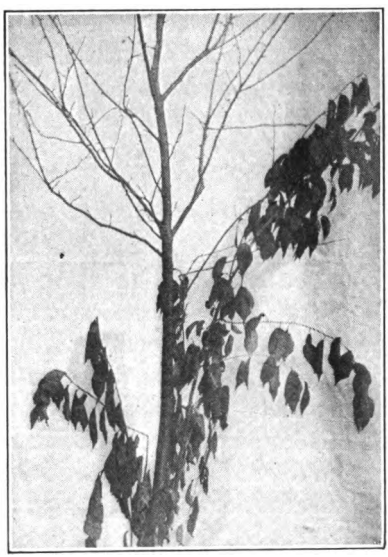

FIG. 111. — Plum-wilt.

From those wilted trees which die in the early summer, gum is exuded. Later beetles bore holes in the bark through which gum flows freely. Death of the tree follows in the fall and winter, and no leaves are put out the next spring. Old trees are said to suffer more than young ones.

Cause.

Recent investigations have shown conclusively that plum-wilt is due to the fungus *Lasiodiplodia Trifloræ*. The pathogene gains entrance into the tree by means of wounds. Observations show that at least 33 per cent of the infections occur through borer wounds. Borers prefer Japanese varieties, probably because they are budded on peach stock in which the borers make their attack. Wounds made by the black-spot pathogene, *Bacterium Pruni*, are also very common points of entrance for the plum-wilt fungus. In this connection it is to be noted that

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Japanese plums are frequently attacked by *Bacterium Pruni*, which fact may, in part, explain their susceptibility to wilt. The wilt fungus may also enter the tree by way of wounds made by cultivating and pruning.

On infecting the plum-tree the fungus spreads out in the bark. Later the medullary rays and sap-tubes are invaded. In the latter the mycelium passes from one duct to another through the pits in the walls. The wood is not destroyed to any noticeable degree. But through the ducts the fungus spreads rapidly; it may pass from a branch into the trunk and thus kill the whole tree. The attacked bark, medullary rays and cambium are killed and finally turn black. During the invasion of the ducts, gum is produced. This partially plugs the vessels and the sap-flow is checked; thus the leaves show a tendency to wilt. Around the margin of an infected area may be found a layer of gum-filled tissue. This sharply limits the fungus for a time. Older trees are not able to limit the fungus in this way, hence they suffer more. Trees eight to ten years old develop gum more readily, yet not in quantities sufficient to prevent rapid spread of the mycelium. While gum stops the fungus temporarily, the deposit in the ducts injures the host. When the attack results in gum deposition throughout a cross-section of a trunk or limb, the affected member dies, apparently from a lack of water.

Control.

From the foregoing the following points should be borne in mind where control measures are necessary: (1) the disease is most serious in Georgia. It occurs also in North Carolina and Alabama. Warmer climates seem to favor it. (2) The causal fungus gets into the tree only through wounds. Of these wounds, those made by the peach-tree borer, the black-spot pathogene, cultivating and pruning are most prominent. Borers and the black-spot organism prefer Japanese plums, because they are budded on peach stock. It is therefore suggested that if some stock other than peach could be satisfactorily

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substituted, much will have been done toward the control of plum-wilt. This line of treatment may offer permanent relief. One should avoid making wounds of any sort. Care should be taken to clean and dress all wounds wherever feasible. Control measures against the black-spot pathogene (*Bacterium Pruni*) are to be emphasized in this connection (see page 311).

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Scab

Caused by *Cladosporium carpophilum* Thüm.

Plum-scab, the same as found on the peach, was observed several years ago on the plum in Iowa. Subsequent observations were made in Delaware, Michigan, Indiana and elsewhere in the United States. It is common in Ontario on wild plums. In Michigan the De Soto variety is said to be particularly sensitive to scab. In Iowa cultivated varieties of *Prunus americana* are reported as susceptible.

As in the case of peach-scab, the chief losses result: (1) on account of marring the appearance of plums; they are unattractive and not of first-grade quality; (2) on account of the cracking which accompanies scab, allowing the brown-rot fungus (*Sclerotinia cinerea*) to enter. The disease is far less common on the plum than on the peach, and in most plum-growing regions it may not be expected to assume dangerous proportions (see page 294 for fuller account).

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ROSETTE

Cause not known

This disease is better known on the peach. Many varieties of plums are said to exhibit the rosette disease, including budded trees and seedlings, cultivated and wild varieties. It has been noted particularly on the Kelsey and Botan varieties in Georgia. With reference to the latter variety, the name has been used synonymously with the Abundance, Berckmans, Red June and Willard, so that it is uncertain which variety is in question. See fuller discussion of rosette under Peach, page 292.

YELLOWS

Cause unknown

This trouble is characterized by the production of wiry yellow shoots as in peach-yellows. The disease is supposedly identical on both the plum and peach, but is by far the more common on the latter. It has been found on Japanese plums and particularly on the variety Abundance. See more detailed discussion under Peach, page 283.

LITTLE-PLUM

Cause unknown

This disease is better known as a peach trouble, but it is said to be quite as common to plums as to peaches in some parts of

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Canada. Japanese plums are more liable to show the disease than European varieties; and it is possible that the latter group is never affected. See Peach, page 289.

Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

Bacterial galls occur on the crown and roots of the plum as in the case of other fruit-trees. These enlargements are less common on this host than on the apple and peach. See Apple, page 108.

Powdery-Mildew

Caused by *Podosphæra Oxyacanthæ* (Fries) De Bary

Sometimes powdery-mildew affects the plum much like it does the cherry, but it is far less common and of no economic importance. See Cherry and Apple, pages 177 and 113.

Armillaria Root-Rot

Caused by *Armillaria mellea* (Fries) Quel.

The plum is susceptible to a rotting of the roots which in many cases is caused by *Armillaria mellea.* In the Pacific Northwest prune-growers have been losing trees on account of this disease for several years. This root-rot is said to do more damage to the prune than to any other fruit in the State of Washington. Likewise in some sections of Oregon, particularly in the northern part of the Willamette Valley, Armillaria root-rot is a very serious menace to the prune industry. For a more detailed discussion, see Apple, page 96.

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Fire-Blight

Caused by *Bacillus amylovorus* (Burr.) Trev.

This is the disease which is so common on pears, apples and quinces in North America. Plums may be regarded as a minor and an occasional host; they are rarely affected. Apparently fire-blight was not observed on plums until July, 1894, although other records of the disease date back nearly a century before. The trouble on plums was first seen in Connecticut, and seven years later (1901) it was noted in Vermont. Recently fire-blight has been recorded on Italian prunes from Oregon. On plum the disease resembles closely twig-blight of pome-fruits. Shoots are suddenly blighted back for a distance of six to eighteen inches from the tip. Full account of fire-blight under Pear, see page 323.

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CHAPTER XIII

QUINCE DISEASES

THE quince is almost always attended by one or two prominent diseases, such as fire-blight and leaf-blight. These two troubles are perhaps the only ones in the United States which should arouse anxiety. Both diseases may be successfully combated. Rust, fruit-rots and crown-gall may also affect the quince.

FIRE-BLIGHT

Caused by *Bacillus amylovorus* (Burr.) Trev.

Fire-blight on the quince (Fig. 112) is no less prevalent than leaf-blight, and it is probable that the former ranks as the most important of quince diseases. It affects orchard trees commonly, and in the nursery is regarded as a very troublesome disease. Entire blocks of quinces in the nursery have been destroyed by fire-blight. The twigs are killed back and on two- and three-year-old stock the blossoms, which occur profusely, are blighted. The disease occurs widely in the United States on the pear, quince and apple, and not infrequently is found affecting the hawthorn, apricot, plum, service-berry and mountain-ash. In the orchard the quince ranks below the pear and apple in susceptibility; however, in the nursery the order of susceptibility is as follows: quince, apple and pear. Among the quinces there are no marked differences in varietal resistance; all

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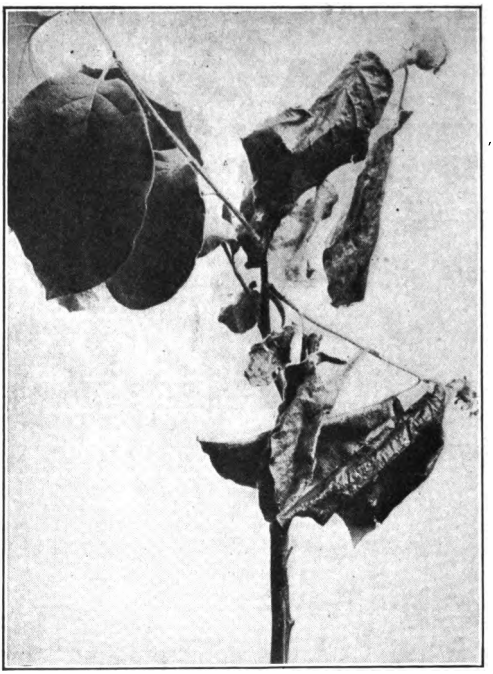

FIG. 112. — Fire-blight on quince.

varieties are susceptible. The fire-blight disease is discussed in detail under Pear (page 323). A special consideration of nursery quinces should supplement the treatment as outlined for fire-blight of the pear. To avoid blossom-blight it is regarded as good practice to remove all blossom-buds from the two-year-old quince trees; this should be done before the flowers open to avoid initial infection.

LEAF-BLIGHT

Caused by *Fabræa maculata* (Lév.) Atk.

This disease occurs also on the pear, to a discussion of which the reader is referred for additional facts and data (page 347). On the quince the leaf-blight disease is also called black-spot, fruit-spot and scald. It was reported from France in 1815, and has a long history in America, now being found in practically all nursery and orchard districts in the Appalachian region. The disease is probably ever present on the quince,

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although it does not necessarily bring about serious difficulty. Yet in nurseries defoliation is common, and sometimes twigs are girdled. The quince orchards in New York State suffer rather severely, especially those in sod or those which are otherwise neglected. Even well-managed orchards are not always free from the disease. All varieties are susceptible.

Symptoms.

Like the spots on pear leaves, there is produced a more or less circular discoloration on the upper surface, with a reddish

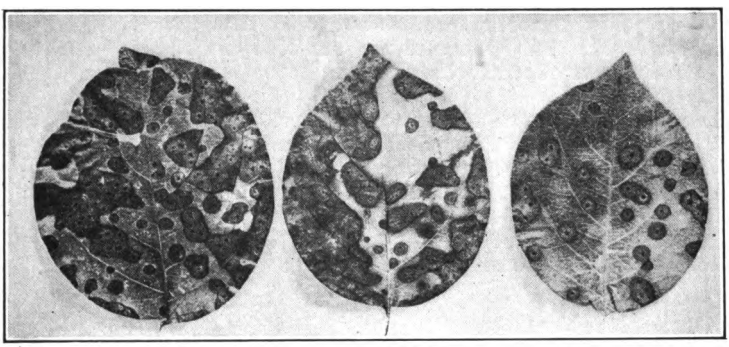

FIG. 113. — Types of lesions on quince-leaves produced by the leaf-blight fungus.

center and dull borders (Fig. 113). Finally the lesion extends through the leaf-tissue showing on the lower surface. The spot on the upper surface becomes dark-brown and a characteristic blackish elevation appears in the center, — the fruiting pustule of the pathogene (Fig. 113). When the spots are numerous, they coalesce (Fig. 113); in cases of severe attack the leaves turn yellow and fall. This defoliation is common in August and September. As a result of a serious infection the quince fruits remain small and the next year's crop suffers. The lesions on the twigs are similar in appearance to those on

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the foliage, with the difference that twig-lesions on quince are more elongated in form and have a depressed surface. The center of the spot shows the fruiting pustule as described for the leaves. On the fruit a black spot is produced. In milder cases the spots are scattered and the effects are not serious; in more intensive attacks the fruits become cracked and lop-sided.

The cause of the disease, as in the case of pear leaf-blight, is the fungus *Fabræa maculata*. For its control see discussion under Pear, page 349.

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See additional references under Pear, page 350.

RUST

Caused by *Gymnosporangium clavipes* Cooke and Peck

Although the damage done by quince-rust is not of great extent, nevertheless the disease has a general prevalence in the region from New England west to northern Michigan, and south to Florida. Outbreaks have been known in Massachusetts in 1897, in western New York in 1910, and the trouble is commonly observed in Alabama, South Carolina, North Carolina, New York, Pennsylvania, New Jersey and Iowa. Generally speaking, quince-rust is not common in the Mississippi Valley. Affected fruits are usually worthless and rusted twigs are considerably malformed.

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Symptoms.

Diseased fruits (Fig. 114) are very conspicuous in July and August. The injured portion is covered wholly or in part by masses of orange fringe-like growths, the whole presenting a yellow woolly appearance (Fig. 114). Some fruits are attacked

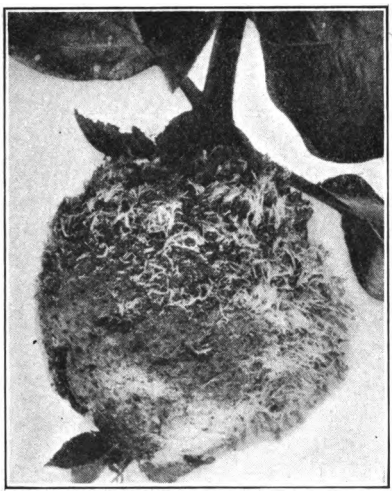

FIG. 114. — Quince-rust.

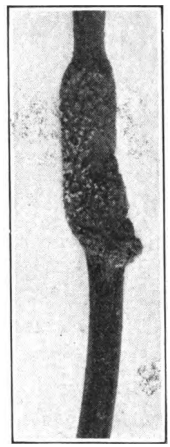

FIG. 115. — Rust on quince-twig.

at the stem end, others at the calyx-end. Again the whole fruit is involved and it may fall. More often, however, it hangs to the tree, dies, and becomes black and hard. There is usually considerable distortion.

Twig-infections (Fig. 115) are commonly found at the base of new shoots, and knots are produced which resemble black-knot of plums.

Cause.

The quince-rust fungus, *Gymnosporangium clavipes*, has a peculiar life-history. A part of its cycle is spent on the quince

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and related plants like the juneberry, hawthorn and apple. But in order to complete the life-history it must have the red cedar, or juniper (*Juniperus communis* and *J. virginiana*). On the red cedar twigs there are produced peculiar canker-like lesions (Fig. 116). These diseased-spots begin their development in midsummer. The fungus hibernates within the twig as mycelium. With the advent of the first warm, moist spring weather the fungus resumes growth and development. On the surface of the affected area numerous yellow, gelatinous masses appear, — the teliospores of the fungus. Each teliospore germinates by developing a short tube (promycelium) on which four sporidia are borne. These sporidia are discharged in thousands, carried by the wind, and some of them infect the quince or the related plants already listed. The sporidia never reinfect the cedars. On the quince, the sporidia germinate, forming germtubes which grow into the tissues of the fruit and twig, causing the distortions and malformations already described. Finally the tubular projections, which in mass are an orange fringe-like growth, develop another sort of spores, — æcio-spores. These are peculiar, like the sporidia developed on the cedar, in that they cannot reinfect the quince, but may infect the cedars. About one year is required for the completion of the life-history of this pathogene.

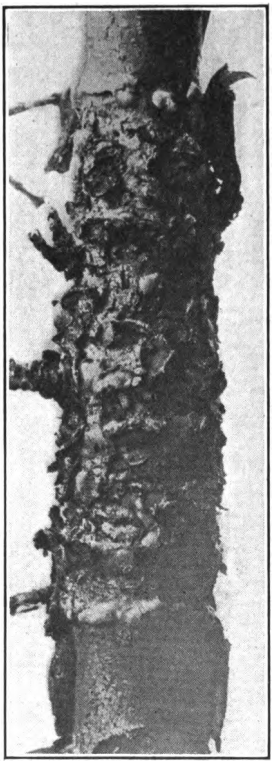

FIG. 116. — Quince-rust on red cedar.

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Control.

The vulnerable point of attack lies in the inability of the fungus to reinfect the quince. It must pass to the cedar. The most direct method of control is to exterminate cedars near the orchard. The juneberry and hawthorn are to be regarded as weeds in the sense that they harbor the undesirable pest, and should therefore be removed. Spraying quinces for the control of rust is not a reliable method of treatment.

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Fruit-Spot

Caused by *Phoma pomi* Passer. = *Cylindrosporium pomi* Brooks

This disease was first noted on the quince in New Jersey in 1892. It was subsequently observed in New Hampshire in 1910 and in Connecticut in 1911. It is now common in New England and in the Middle Atlantic region. The Orange quince seems particularly susceptible. The disease also affects the apple and Chinese quince.

Fruit-spot, or blotch, appears late in August or early in

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September. Lesions show anywhere on the surface of the fruit, but are most numerous near the blossom-end. Here they frequently unite, forming one continuous blotch. The spots are one-fifth of an inch or less in diameter, their surface is slightly sunken, and the affected tissue firm. At first the diseased portion is a deeper green than the surrounding tissue. Later the lesions are black. In the center of each spot black specks — fruiting bodies of the pathogene — appear in great numbers. See fuller discussion of this disease under Apple, page 87.

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BLACK-ROT

Caused by *Physalospora Cydoniæ* Arnaud

The disease which is discussed under Apple (page 45) occurs on all pomaceous fruits, including the quince. The trouble manifests itself on quince almost entirely on the fruit and is referred to as black-rot. The foliage and woody parts of quince are rarely affected to any extent; consequently the leaf-spot and canker forms of this disease are not given consideration in this discussion.

Black-rot of quince was first observed in Connecticut about 1890, when it was prophesied that it might become a serious menace to quince-culture. While its range over the northeastern United States is general, and its occurrence in and about Ohio and New Jersey is common, yet the disease ranks as one of minor importance on the quince. The disease is well known and destructive on the apple.

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Black-rot makes its appearance on green fruit in August and may be in evidence until the fruit is picked. A lesion begins as a brownish speck. It may be located anywhere on the surface of the fruit, but the blossom-end commonly marks the center of a diseased area. As the size of the spot increases, the color darkens, the affected skin wrinkles, and black pimples may appear over the surface. Sometimes these bodies do not show until the whole fruit is involved; this is usually a matter of two or three weeks. Finally the fruit is completely mummified and after drying is no more than one-fourth its normal size.

See fuller discussion under Apple, page 45.

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BITTER-ROT

Caused by *Glomerella cingulata* (Stoneman) Sp. and von S.

Where this disease prevails it is quite destructive. However, its range and frequency are not sufficient to render it of an alarming nature. Quinces affected with bitter-rot show symptoms very similar to those exhibited by the apple, on which fruit this disease is so common in warmer states. Affected portions on the quince turn brown, the surface becomes depressed, and there appear the characteristic pinkish dots over the lesion. The name implies that the rotten flesh has a bitter taste; however, this is not a constant character. On this account, the name ripe-rot has been suggested as a substitute. But the rot is not entirely a disease of ripe fruits, hence ripe-

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rot is fully as objectionable. Usage demands that the name bitter-rot be employed. Quinces grown in close proximity to apples affected with bitter-rot may be expected to succumb. However, if history is a reliable basis for prediction, the disease may never be expected to assume destructive proportions. (See Apple, page 14.)

REFERENCE

Halsted, B. D. Some fungous diseases of the quince fruit. The ripe rot of quinces. New Jersey Agr. Exp. Sta. Bul. 91 : 11–12. 1892.

BROWN-ROT

Caused by *Sclerotinia cinerea* (Bon.) Schröt.

The decay of quince fruit caused by *Sclerotinia cinerea* is distributed generally over the globe. A closely related species of fungus (*S. fructigena*) very frequently attacks apples and pears as well as quinces in Europe. Considerably less damage is done to the quince than to apples and pears. It is also less destructive in America than in Europe on any of the three above-named fruits. Sometimes *S. fructigena* attacks stone-fruits in Europe; however, the brown-rot of stone-fruits and pome-fruits in America is due chiefly or entirely to *S. cinerea*. The characters of brown-rot on quinces are not notably different from those described for peaches, to which the reader is referred (page 270). Special control measures do not seem necessary, at least in America.

CROWN-GALL

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

Galls in the form of small swellings occur on various portions of the limbs and twigs of quince trees. Often an entire limb is covered by the irregular, warty outgrowths. Crown-gall is

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the name applied to the disease irrespective of the location of the galls on the tree. The same disease affects the apple, peach and other plants. Pacific Coast growers meet with this trouble more than others in the United States. The disease also occurs in Africa. (See Apple, page 108.)

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(Other references, page 112.)

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CHAPTER XIV

RASPBERRY DISEASES

Not infrequently raspberry diseases have been factors in driving the bush-fruit growers out of the business. Crown-gall is very destructive to raspberries, although its injury to trees like the apple and peach is questioned. Orange rust is very damaging to black-caps, and yellows is extremely common everywhere. Anthracnose, cane-blight and root-rot are troublesome in many localities where this fruit is grown.

Crown-Gall

Caused by *Bacterium tumefaciens* E. F. Smith and Townsend

Note should be taken of the fact that crown-gall occurs on a great variety of plants (see list on page 112). In certain states, for example New York, the trouble affects the raspberry more commonly than any other plant. It is also more destructive to the raspberry, in many cases, than to the apple, peach, grape or other fruits. While no variety appears immune, the disease is particularly common and important on the red varieties like the Cuthbert and London. It is said to cause considerable damage in the State of Washington; and records of it come from several other states indicating its wide geographical range in the United States. It was observed in Ohio as early as 1894.

A more detailed discussion of crown-gall will be found under Apple, page 108.

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ORANGE-RUST

Caused by *Gymnoconia interstitialis* (Schl.) Lag.

This is a well-known raspberry and blackberry disease over the eastern United States and southern Canada. It also occurs as far south as Florida and California. The disease is found in Europe and Asia commonly.

The attacks are limited to wild and cultivated blackberries and raspberries. It is extremely common on black raspberries in New York. In Illinois the Snyder is said to be resistant. On all of these forms the disease is variously referred to as orange-rust, spring-rust, red-rust, bramble-rust, and erroneously as yellows. The disease destroys the usefulness of the leaves and they finally fall. Annual recurrence of this phenomenon results in rendering the plants worthless. Rarely do affected plants recover. Ten per cent rusty plants are frequently reported. Twenty-five per cent or more are recorded. In some localities the orange rust has so seriously affected raspberries and blackberries as to make their cultivation unprofitable.

Symptoms.

First signs of the disease are observed in the spring — whence the name spring-rust. In April and May, even before the leaves are entirely unfolded, evidences of the disease may be seen. On the upper surface of the leaves glandular bodies are

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developed. In their full maturity they appear as black specks. The tissue about them is yellowish. These bodies are not found on all leaves nor on all leaflets of a given leaf. Two or three weeks after the appearance of these structures evidence of rust is seen on the lower surface of the leaves. When mature, these bodies (sori) break open and expose an orange-colored mass of spores; whence the name orange-rust. Ordinarily these sori with their spore-masses practically cover the lower surface of the leaf. Affected leaves are dwarfed and rolled, somewhat exposing the orange-colored lower surfaces. Rust is rarely found on the canes. This stage gradually disappears during the latter part of June, until by July it is difficult to find in northern United States. Farther south, on a parallel with Maryland, orange-rust may be seen in late summer and sometimes in the fall. Affected plants are noticeably stunted (Fig. 117) but are not killed. Plants once affected are almost certain to be diseased again the following year. Some rusted plants show a reduced number of prickles.

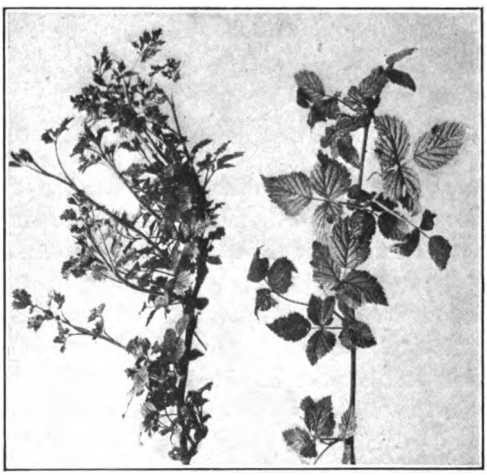

FIG. 117. — Orange-rust; healthy plant on right, diseased on left.

Cause.

The bramble fungus, *Gymnoconia interstitialis*, is the cause of this disease. Its mycelium lives from year to year in the

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affected plant. In the roots the mycelium is found between the cells of the cortex, near the cambium. As it proceeds upward the pith of the canes is invaded. In the leaves the spongy parenchyma is infected. In young stems the hyphæ may be found in any of the tissues. In all these attacked organs the mycelium develops haustoria. These arise as side branches from the mycelium. They pierce the cells to get food. As an infected cane grows the fungus follows the growing-tip. In the spring on the upper surface of the opening leaves sterile black bodies appear. These are called spermagonia, or pycnia. Two or three weeks later the orange-colored cushions develop on the lower side of the leaf. Many spores are formed, which are capable of germinating at once. From each spore a short promycelium bearing four sporidia is protruded. These sporidia cause other infections on raspberries and blackberries during the growing-season. Mycelium develops from the sporidia. In the winter the fungus is again dormant as mycelium in the canes, crown and roots.

Control.

The perennial nature of this rust fungus makes it difficult to control. The only known remedy for diseased plants is to dig and destroy them. All wild blackberries and raspberries known to be affected should be destroyed; they may be regarded as weeds in this connection. Spraying to protect healthy plants from infection should be beneficial, although no experimental data on which to base specific recommendations are available. The fungus has definite enemies which destroy the spores in large numbers. Certain insects eat quantities of spores. A certain fungus, *Tuberculina persicina*, is parasitic on the rust pathogene.

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YELLOWS

Cause unknown

This peculiar and obscure raspberry disease is variously known as curl, leaf-curl, yellows and the Marlboro disease. The term yellows is used to cover a variety of symptoms and is very loosely applied by the grower to any condition in which the leaves turn yellow. The name Marlboro disease is local in usage; it owes its origin to the fact that the Marlboro variety is very susceptible. By reason of its descriptive nature the name yellows seems most appropriate.

Raspberry-yellows was first recorded from Minnesota in 1894. At present its geographical range is probably coincident with the plant affected, having been observed at one time or another in Ohio, New York, Connecticut, Massachusetts, Pennsylvania, Kansas, Michigan, Colorado, California and Washington. It is also said to occur in Canada. All who are acquainted with the disease concur in that it is very important. In certain localities it is regarded as the most prevalent and the most destructive of raspberry diseases. In many cases where the raspberry industry has been ruined the yellows disease has been held responsible for such conditions. Red raspberries stand alone in their susceptibility to injury from yellows. Only rarely are the black-caps and purple varieties affected appreciably. In order of their susceptibility red raspberries follow:

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Cuthbert, Marlboro, Golden Queen, Early King and Herbert. The St. Regis is resistant. Occasionally other red varieties are affected. Often the Cuthbert and Marlboro are so injured that they are discarded from commercial plantations. Where the disease has wrought noticeable damage the original acreage has been reduced to one-fifth within a period of eight years. In such localities the annual loss is estimated at $200 per acre.

Symptoms.

The true raspberry-yellows (Fig. 118) has a striking and characteristic appearance and should never be confused with

FIG. 118. — Raspberry-yellows; healthy plant on left, diseased on right.

lack of thrift in plants due to cultural conditions, nor should it be confounded with cane-blight. A withering or blighting of canes or leaves does not occur in the case of yellows. In general the disease does not appear until two years after planting; sometimes three years may elapse before it really attracts

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attention. Once affected, the plant continues to show signs of yellows annually to some extent. Cases are on record where recurrence of the disease has taken place for nine consecutive years.

Both healthy and diseased raspberry plants may occur in the same stool. In extended outbreaks all gradations between healthy and affected plants obtain. One of the more striking symptoms of yellows is that the plants are stunted, sickly and bushy in aspect (Fig. 118). In general the condition recalls that of peach-yellows. The fruit-bearing laterals are dwarfed, often being but one-half their normal length. The leaves themselves present an unmistakable characteristic when affected with yellows (Fig. 118). They are abnormally small, and the margins of the upper leaves curl downward (Fig. 118). The tissue between the leaf-veins arches upward, and as a result the veins appear sunken. This uneven growth brings about a curling of the foliage (Fig. 118). In summer, the foliage acquires a mottled appearance (Fig. 118); at first a light coloration prevails, then it gradually changes to a darker green and yellow, and finally is reddish bronze. The colorations accompanying and symptomatic of yellows vary considerably with the soil and climatic conditions. The disease is more conspicuous in dry and hot weather. The berries on diseased plants usually become dry before maturity, or they ripen ten to fourteen days early. In case they dry up, the change may be gradual or sudden. If the fruit ripens prematurely, it is bitter, lighter in color, and smaller than normal. Not uncommonly the flowers develop prematurely on the tip of the current year's growth. From all appearances the root-system is normal.

Cause.

As yet the causal nature of raspberry-yellows is unknown. Various factors have been assigned in this connection, but always without proof. Among these may be noted fungi, insects, bacteria, poor drainage, lack of soil fertility and other

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similar factors. Authorities agree that fungi cannot be the cause. Likewise entomologists say that insects are not concerned. The red spider has been suspicioned, but conclusive evidence is lacking. If bacteria are the cause, they have not been seen. It is possible that they are present, but are so very small that they are easily overlooked. It has been noticed that the disease is more pronounced where plants were growing in soil which had a high water table. Plants growing on soil having compactness and lacking drainage are more liable to exhibit yellows than lighter well-drained soils. It has been suspicioned as having some connection with crown-gall and cane-blight, but careful observations do not substantiate this view.

Control.

No preventive measures are known, and little hope is in sight until the causal nature is understood. Fungicides have proved themselves worthless in controlling the disease. A few recommendations have been suggested, and the more important of these may be noted: (1) in planting red raspberries obtain plants from localities where raspberry-yellows is known not to occur; (2) select varieties which have shown resistance in the neighborhood; (3) select for a plantation-site a light, or medium-heavy, adequately drained soil; (4) irrigation is helpful where practiced; (5) destroy diseased plants.

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ANTHRACNOSE

Caused by *Glœosporium venetum* Speg.

The disease under consideration is generally known as anthracnose, which name was given it in 1887. Prior to that date it was called cane-rust. The first account of the disease comes from Illinois dated 1882. Complaints were made about 1885 to 1887 of the serious injury which anthracnose was doing in Illinois, Wisconsin, Missouri, Texas and New Jersey. The sources and number of these complaints indicated that the disease was then widespread and destructive. More recent records of the trouble show that it occurs in the North Central States, the North Atlantic Division and the Western Division where the output in quarts is greatest.

Raspberry-anthracnose (Fig. 119) also affects the blackberry (see page 161). The raspberry, however, suffers more from this disease than the blackberry. All above-ground parts are injured more or less. Canes are sometimes girdled and are therefore killed. Leaves are about one-half their normal size and distorted when affected by anthracnose. Fruits, if formed at all, may not reach their full development, but they ripen prematurely and are therefore worthless. If young canes are not killed the first year, the formation of fruit for the next year is prevented.

It is difficult to estimate the amount of the losses incurred from anthracnose because of the nature of the losses. It has been estimated in Missouri that 10 to 12 per cent of the entire crop is injured. In 1904 in one county in Nebraska 33 per cent of the crop was injured, and in 1907 one-half the crop in Wisconsin is thought to have suffered. But no records based on carefully selected data are available which will indicate the annual dollar loss. In many plantations everywhere the disease is enphytotic, the amount of the injury being about the

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same from year to year and in no case disastrous or fatal to the affected plants.

Some varieties suffer more than others. In general, black raspberries are more susceptible than red varieties. Among the red raspberries the Antwerp is injured to a considerable degree, and the spots are very conspicuous thereon. The Cuthbert, on the other hand, is but slightly affected. Among the black varieties the Cumberland is susceptible.

Symptoms.

Although anthracnose is preeminently a disease of the canes, no part above the ground is free from the attacks of the pathogene. The disease first appears on the canes when they are from six to ten inches high. Both fruiting and non-fruiting canes (suckers) are affected. On the suckers the disease shows more commonly at the base. In any case the larger and older lesions are found toward the base of the cane. The course of development appears to take place from below upwards. Younger and smaller lesions always are to be found toward the tip of the cane. The spots (Fig. 119) are at first small, purplish and variously scattered. Their form is elliptical. As the lesions become older they increase in size and the color, particularly at the center, becomes grayish white (Fig. 119). The margin remains purplish. It is slightly raised, and thus the healthy and diseased tissues are sharply separated (Fig. 119). In more advanced stages the affected areas coalesce and the cane appears irregularly blotched. These blotches are often an inch and a half long and sometimes girdle the cane. The affected cane attempts to heal the wound so that old spots have a rough, scabby aspect. Sometimes canes are severely cracked (Fig. 119), in which cases the pith is exposed. Often the affected areas present an appearance of large irregular cankers in which the surface is sunken and the bark split lengthwise. Canes thus affected cannot function properly, and as a result they become sickly. Their leaves are dwarfed and the fruit,

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if formed at all, never reaches full maturity, but ripens prematurely or dries up.

The petioles of the oldest leaves are attacked soon after the canes are affected. Purplish spots as described for the canes above are developed. The affected tissue enlarges along the petiole, finally reaching the leaf proper. On the leaf whitish, blister-like lesions appear. The leaves then become distorted, their edges rolling in toward the midrib. Spots on the leaf-blade are similar to those on the canes, although smaller; they are one-twenty-fifth of an inch or less in diameter. They are closely distributed, but rarely coalesce. Frequently the diseased area, in which most of the tissue is dead, becomes separated from the surrounding healthy tissue and the leaf shows a shot-hole effect. Leaves on the laterals of new canes do not ordinarily become diseased.

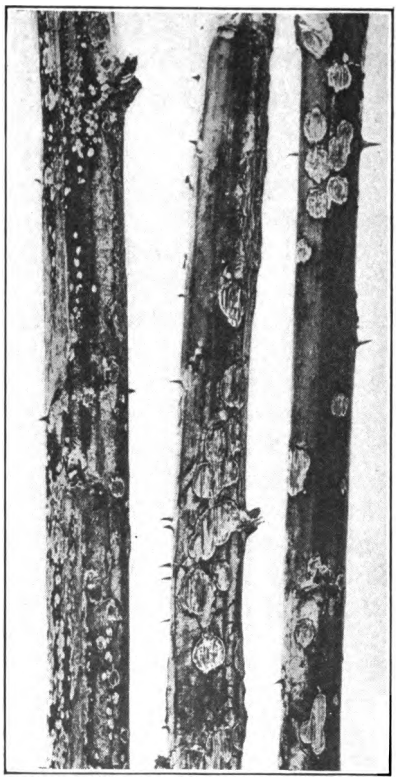

FIG. 119. — Anthracnose on canes of black raspberry.

Anthracnose shows on the fruit. The upper drupels are more commonly attacked. Many fruits are affected while still green in color and sometimes before they are larger than a pea. A small brown

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speck develops on the end, but soon the entire surface is involved. As the fruit matures the surface of the lesion becomes depressed. If affected while small, the drupels remain firm and finally become dry. Fruit-pedicels may be attacked, in which case the fruit dries up and dies.

Cause.

The anthracnose lesions just described are caused by the fungus *Glœosporium venetum*. Details of its full life-history are as yet unpublished, but the facts as generally known will be discussed. The winter is spent in the canes. It is very likely that in addition to mycelium, immature fruiting structures are concerned in this connection. In the spring conidia are formed on the old diseased areas by the fungus. These spores are disseminated about the time the plants are from six to ten inches high. Apparently the exact origin of the conidia is not generally known. The perithecial stage has recently been described, but as yet is not named. The ascospores developed within the perithecia are shot forcibly into the air and later germinate by budding. These buds are identical with the conidia; they may be referred to as such. Conidia probably also are developed in acervuli. These spores attack and their germtubes penetrate only the tender growing parts of the cane, as evidenced by the smaller and younger spots at the tip and the older ones at the base (see Symptoms). Following the germination and penetration processes mycelium ramifies in the cortex of the cane where the cells are killed. Subsequently conidial fruit-bodies are developed from which conidia are liberated. These initiate secondary infections on the canes, leaves and fruits. These secondary infections continue throughout the summer. In the winter the fungus again remains dormant in the canes.

Control.

Diseased canes should be eradicated before the spring season. It is preferable that this be done at the end of the fruiting-sea-

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son. Short rotation should be practiced. Ordinarily it will not pay to keep a plantation of black raspberries after it has produced its third crop. Clean, anthracnose-free plants should be set. All weeds should be kept down; this operation tends to reduce the relative humidity about the canes; moisture favors the fungus.

It is possible to keep a plantation relatively free from the disease by the use of bordeaux mixture 4–4–50 in the spring. The applications should be made as follows: (1) before the leaves appear; (2) when the leaves are well developed and by the time the shoots are six inches high; (3) just before the blossoms appear. Whether subsequent applications are needed will depend on the weather and the severity of the disease. Later sprayings are made at ten-day intervals.

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Cane-Blight

Caused by *Leptosphæria Coniothyrium* (Fckl.) Sacc.

This disease was first observed in 1899 in the Hudson Valley, State of New York. Until that time it was wholly unknown to science, although it was then abundant and destructive.

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Later reports indicate that raspberry cane-blight now has a very general range in the plantations throughout New York State. Its geographical distribution over the United States is not well known, but it is doubtless common throughout the country. It has been seen in Ohio, Wisconsin, Connecticut and a few other states.

The chief damage is done to the fruiting canes. New canes are attacked, however, and occasionally are killed during the first season of their growth. This is not the most important raspberry disease generally, and rarely is a whole crop lost. On the other hand, few plantations are entirely free from it, in New York at least, and cases are on record where one-fourth to two-thirds of the crop was lost through the effects of cane-blight. It may easily be imagined that the aggregate losses in the country must be considerable. It is said that cane-blight is often partly responsible for the early decline in the productivity of both the red and black raspberry plants. Death of the affected canes at the point of attack results in a wilting of both the cane and its foliage. Hence, the disease is sometimes called raspberry cane-wilt. In such cases the berries become dry and worthless. In many instances the berries are attacked directly, resulting in a dry rot.

The disease affects nearly all of the red and black varieties, and perhaps also the dewberry and wild red raspberry (*Rubus strigosus*). There is nothing to indicate the occurrence of cane-blight on the blackberry, except on wild species. Among the commercial raspberries, the Cuthbert variety probably suffers most. Other varieties, such as the Marlboro, Ohio, Gregg, Kansas, Superlative, Pride of Geneva, Parmer and Cumberland, are affected considerably. The Columbian is notably resistant.

Symptoms.

Observations indicate that the disease may be expected to appear in neglected and well-managed plantations alike. The

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lesions are found most commonly on the canes (Fig. 120), although the berries may be directly affected. All or only a portion of a cane is involved, in which case the foliage suddenly wilts and becomes dry. Often only a single branch wilts; the remainder of the plant then continues normal activities and appearances (Fig. 120). Frequently only a portion of a cane is blighted, such symptoms becoming evident as soon as the leaves unfold in the spring. Lesions commonly center about a wound left in pruning, from which point they extend downward. There is a tendency on black varieties for the disease to affect only one side of a cane. The diseased area is brown and the cane becomes very brittle at such points. Very early in the blighting of a cane, black fruit-pustules of the pathogene appear on the lesions (Fig. 121); from these pustules masses of reproductive bodies ooze out on the bark, giving the affected portion a brownish smoke-colored appearance. The spots are not always limited in extent; in some cases they are generalized, the wood cracks, and the bark peels off at the lower portion of the affected cane.

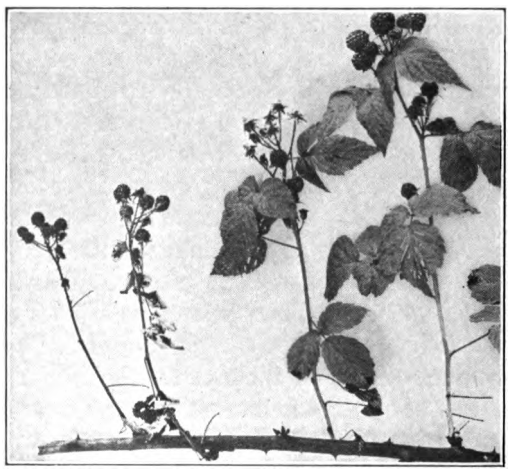

FIG. 120. — Raspberry cane-blight; healthy plants on right, blighted canes on left.

The cane-blight disease may be confused with the work of the raspberry-cane borer (*Oberea bimaculata*). But in cane-

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blight no insect burrows are found. The disease has also been confounded with drought and winter-injury. Drought-injured canes dry up slowly and more uniformly; blighted canes die suddenly. Winter-injured canes do not put out new leaves on the affected portion; blighted canes develop new leaves on the affected parts.

The berries dry up as a result of cane-wilt (Fig. 120). They are also susceptible to direct attack. This is evidenced by the fact that a single berry in a cluster, or even one side of a berry, may be diseased. The normal green color is slightly tinted as if ripening prematurely; finally, the tissues gradually turn brown and a dry rot results.

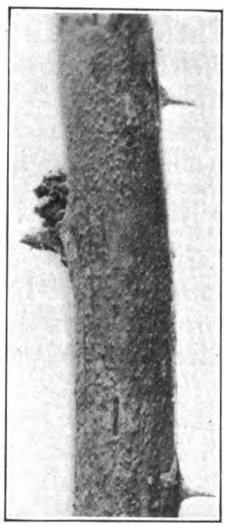

FIG. 121. — Cane-blight; fruit bodies of the pathogene on raspberry cane.

Cause.

The brown mass of reproductive bodies, already mentioned, which ooze out on the affected bark are the conidia of the causal fungus, *Leptosphæria Coniothyrium*. These spores are disseminated from plant to plant probably by insects (tree crickets), wind and dashing rain, by pickers, pruners and cultivating tools. The spores germinate on the canes, and evidently the germtubes are capable of penetrating the unbroken epidermis of the canes. The fungus also enters the canes through wounds; stubs left exposed in heading-back and injuries made by the snowy tree cricket (*Œcanthus niveus*) are common points of entrance. Berry-infection doubtless occurs through the flowers and very young fruit. The mycelium works between and within the fleshy parts of the drupelets, but not in the embryo or stony part. It passes from the fruit down the pedicels and thence upward to other berries of

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the cluster. The fungus evidently hibernates as pycnospores in pycnidia; in the spring these spores ooze out on the bark. Dead and decaying canes are sources of the inoculum. It has been shown that the fungus may live as long as four years on fallen dead canes. It is believed that the pathogene is carried in the soil particles on nursery-stock. Furthermore, the practice of laying down and covering of canes with soil in the fall to protect them from winter-injury, is thought to spread the organism. A perithecial stage has been discovered, but its rôle is unknown.

It is peculiar that the fungus may cause most serious damage in dry seasons. This is explained on the grounds that canes become infected during the first season of their growth, but do not show effects until the following year. Weather conditions have a positive influence on inoculation and incubation, but not on the growth of the fungus in the cane.

Control.

No definitely established remedial measures are at hand. Ordinarily plantations may be expected to recover, even if badly diseased for one year. As a precautionary measure, plants should not be selected from nurseries in which the cane-blight is present to any great extent. Since the fungus may live on fallen canes for four years, it is inadvisable to replant on soil within this time after a severe attack. In any case the old canes should be burned. Dead and diseased canes should be removed soon after the fruiting-season, and again in the early spring. If the disease is particularly troublesome, look for any wild blackberries and wild red raspberries in the vicinity of the plantation. Their destruction is one step toward eliminating the fungus.

According to present knowledge, the application of fungicides is not promising. In some cases bordeaux mixture has been applied as follows: (1) when the canes are a few inches high; (2) subsequently at intervals of two weeks until the middle of

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September; and (3) again the following spring from the bursting of the buds to the setting of the fruit. Future experience may prove spraying a more beneficial measure of control.

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Leaf-Spot

Caused by *Septoria Rubi* Westd.

This disease is less troublesome on raspberries than on blackberries and dewberries. It has been reported from several eastern and middle western states, but no cases of severe injury are recorded.

The spots often appear as early as the middle of June in the form of whitish or faintly brownish areas. Frequently they are numerous, and in very severe cases an affected leaf dries up. In rare cases this results in the development of inferior fruit which is small, dry and tasteless.

The disease is controlled by the use of bordeaux mixture, although the foliage of raspberries is delicate, and susceptible to injury by this fungicide. (See Blackberry, page 166.)

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Armillaria Root-Rot

Caused by *Armillaria mellea* (Fries) Quel.

Raspberries are seriously injured by the shoe-string fungus in the Pacific Northwest. (See fuller account under Apple, page 96.)

Blue-Stem

Caused by *Acrostolagmus caulophagus* Lawrence

This is a raspberry and blackberry disease peculiar to the Pacific Northwest. It has been increasingly troublesome in the Puget Sound region since 1904. All blackcap varieties of raspberries are susceptible. It also affects red raspberries.

Symptoms.

In general, when raspberries are affected with blue-stem the plants fail to mature the crop properly and they may die during the summer. The canes usually are first to show signs of the disease. The fruit begins to dry up and harden before maturity. This change in the fruit is accompanied by a discoloration and wilting of the leaves and a darkening of the canes. Symptoms vary with the severity of the attack.

The first discoloration of the shoots occurs a few inches above the ground. A blue-black stripe extends from some point a few inches above the ground upwards on the shoot. The stripe may be narrow, or the whole side of the shoot may be dark-blue. Very commonly shoots are discolored throughout their circumference for three feet from the ground. This discoloration may increase three to six inches in length each day during the growing season. At the ends the dark-blue area is fringed and fades into a reddish brown color. The margin is quite distinct. As a result of this affectation some shoots may die. Death occurs throughout the entire growing season, al-

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though most dying takes place during the first half of the summer. Other plants which do not die show a wilted and yellowed condition of the foliage. Canes do not always show external signs of the blue-black discoloration. Internally, however, affected canes exhibit characteristic reddish streaks. The roots also may show this internal discoloration. In a single plantation every hill may show the blue-stem disease.

Cause.

The life-history of the causal fungus, *Acrostolagmus caulophagus*, is not fully known. It apparently lives in the soil and in some way gets into the plant at a point below the surface of the ground. There is some evidence that entrance is gained through the roots. The mycelium invades the wood ducts profusely. Progress in the roots is slow; however, in above-ground parts the rate of spread, as already indicated, may be from three to six inches each day. The mycelium at times completely plugs the wood-ducts, thus accounting for the wilting, yellowing and death of affected plants.

Control.

As yet no experimental data are at hand on which to base reliable recommendations for the prevention of blue-stem.

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SPUR-BLIGHT

Caused by *Mycosphærella Rubina* (Pk.) Jacz.

This disease has been reported only from Colorado and New York. It probably occurs elsewhere. Both black and red varieties of raspberries are affected. In Colorado it is regarded as a very important trouble. It ranks well up with other factors

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responsible for marked decrease in production such as late frosts, poor management and age of plantations. It reduces the crop by reducing the number of fruit-spurs. Buds are killed and leaves may die. From 2 to 50 per cent of the canes are lost through their brittleness; for when brittle they break easily while being put down in the fall or taken up in the spring. In New York the disease is far less injurious than in Colorado.

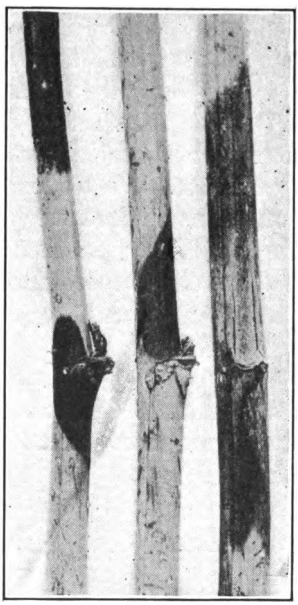

FIG. 122. — Spur-blight of raspberry.

Symptoms.

The disease is first apparent in July. The lesions, one to several on a cane, are sharply defined, conspicuous, and measure from one to four inches long and may half-way girdle the cane (Fig. 122). Spots are found on the nodes near the ground. They may also occur on the internodes and petioles of the leaves. An area about the buds at the base of the leaves becomes brown, the buds shrivel and become dry. The buds then are either weakened considerably, so that they make only feeble growth the following spring, or they die. In the former case the buds come out, but are small, soon turn yellow, and die. On account of this disease fruit-spurs may not be found for two feet above the ground. However, berries are produced on the upper half. Lesions on the nodes and internodes may coalesce so that the whole lower portion of a cane may become dark. The affected bark splits longitudinally, thus allowing the cane to dry out prematurely. As a result of this desiccation the cane is more brittle than

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normal. The lesions become whitish at the center in September and October. Later small black pustules develop.

Cause.

The fungus *Mycosphærella Rubina* causes spur-blight. Its mycelium invades the cortex, wood and pith. Within two weeks after the fungus falls on the spur a typical lesion is produced. The fungus apparently hibernates as immature perithecia in the canes. In the spring these bodies mature their ascospores. This occurs about the first of May. These spores are liberated and are carried by the wind to young canes which are at this time but a few inches high.

Control.

It has been demonstrated in Colorado that spraying will prevent spur-blight. The time of inoculation necessitates that spraying be done early in the spring. Bordeaux mixture 3–2–50, with the addition of resin-fish-oil-soap at the rate of two pounds to fifty gallons of bordeaux as a sticker, is advised. Apply to the young canes only, and care should be taken to coat the portion nearer the ground. Four applications are advised; three before picking at intervals of two weeks and one immediately after picking. The first application should be made when the plants are only a few (eight to twelve) inches high. This is the last of May in Colorado. The second spraying follows two weeks later. The third two weeks after the second. Old fruiting canes should be removed and burned immediately after harvest to prevent infection of new canes.

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CHAPTER XV

STRAWBERRY DISEASES

STRAWBERRY-GROWING is often handicapped by pests of one sort or another. Among these the leaf-spot and powdery-mildew, and at times the Botrytis-rot, are important. Growers of strawberries do not rely on spraying alone for the control of their strawberry enemies, but practice sanitation, rotation and cultivation. These measures of eradication are, of course, to be supplemented by protecting the plants with some good fungicide.

LEAF-SPOT

Caused by *Mycosphærella Fragariæ* (Schweinitz) Lindau

Perhaps the most common of strawberry diseases is the leaf-spot. It is also called spot disease, sun-burn, sun-scald, leaf-blight, and, erroneously, strawberry rust. These names will recall to the mind of the reader the disease under consideration.

This leaf-spot is found in Europe and America. First studies were made in France in 1865. About 1880 it began to attract more than usual attention in the United States, and for five or six years thereafter complaints of its prevalence and destructiveness came from many parts of this country. While the trouble is now known in practically all parts of the United States, the strawberry-growers of the northeastern quarter of the country are the heaviest losers on account of this disease.

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The disease is usually less severe in Florida, Louisiana and other southern states.

In many cases the amount of injury done by the leaf-spot is doubtless over-estimated. Yet it must be remembered that the disease does some damage nearly every year, so that after all the pest is not only one of the most common but also one of the most destructive to strawberries. In certain localities it is of comparatively slight import, while in other places whole plantations have been completely destroyed. In southern Illinois, a leading strawberry section, leaf-spot is regarded as one of the worst enemies of this fruit. In Connecticut, one grower is said to have experienced a loss of $250 an acre, while the damage done in 1904 to Maryland strawberries is estimated at 21 per cent of the total acreage. This approximates a loss of $50,000.

In the ordinary cases of attack the foliage is impaired; in severe cases the leaves are affected to the extent that the plants die. In any case, the prospect for the next year's crop is usually threatened. If the fruit-pedicels are attacked, the berries never mature but remain small, become shriveled and worthless.

The most serious injury is said to occur on such varieties as the Wilson, Hunn, Beeder Wood, Warfield, Sanafee, Photo, Monmouth and Ohio. The varieties which resist the disease to a noticeable degree are the Marshall, Brandywine, Lady Thompson, Michel's Early, Daisy, Eureka and Jewell. It is not guaranteed, however, that the resistance and susceptibility herein indicated will be found applicable or reliable in all strawberry sections. It is well to bear in mind that ecologic factors may have no little influence in this respect. Accordingly, then, observations as to varietal resistance should be carefully considered. It is generally held that vigorous plants are no more resistant than the more feeble individuals. It has also been observed that plants growing on heavy, wet, undrained

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land are more severely diseased than those on light, well-drained soil. Plants exposed to the sun are said to be more affected than those which are growing under shade; it appears that shade has a preventive effect, a phenomenon not easily explained. Moisture alone increases the amount of leaf-spot, yet during an excessively rainy season plants remain quite free from the disease. This freedom from leaf-spot during a very wet season has been explained on the grounds that cloudy weather offers much the same conditions as those of shade. In a year of continued drought the disease is likewise less common. In general, conditions most favorable to the disease are hot, bright days with occasional showers or heavy dews.

Symptoms.

The disease makes its appearance on the leaves (Fig. 123), calyx and fruit-pedicels. In the beginning, the spots are very small, deep purple or red, and are usually first evident on the upper surface of the leaves. These areas rapidly increase in size, and at the same time the color of the central portion changes from a purple to a reddish brown; eventually the lesions become grayish or whitish in the center (Fig. 123). The border remains purplish, shading off to a reddish brown coloration towards the healthy. The single lesions vary from $\frac{1}{8}$ to $\frac{1}{4}$ of an inch in diameter, but several spots may coalesce to form a large irregular blotch. Severely affected leaves turn brown, this discoloration beginning at their tips; finally such leaves shrivel and die. The lesions on the other susceptible organs are similar to those on the leaves.

Cause.

The leaf-spot disease of strawberries is produced by the fungus *Mycosphærella Fragariæ*. The mycelium of the fungus grows in and between the cells, and through the action of these threads upon the host-cell contents there is a response expressed in the spots as described. After a short time the hyphæ

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amass themselves beneath the cuticle of either surface of the leaf, and from these masses conidiophores bearing conidia arise. These spores develop, are scattered and produce infections throughout the summer; with the advent of winter conidial formation ceases, but the mycelium remains alive in the leaves until the following spring. At this time a new crop of conidia develop, which begin the cycle anew by infecting again the strawberry plants. Should any of these spores perchance fall on the common cinquefoil, the disease would be produced. It should also be mentioned that this plant acts as a weed-host, carrying the fungus through the winter and furnishing a source of the inoculum in the spring, as does the strawberry plant itself.

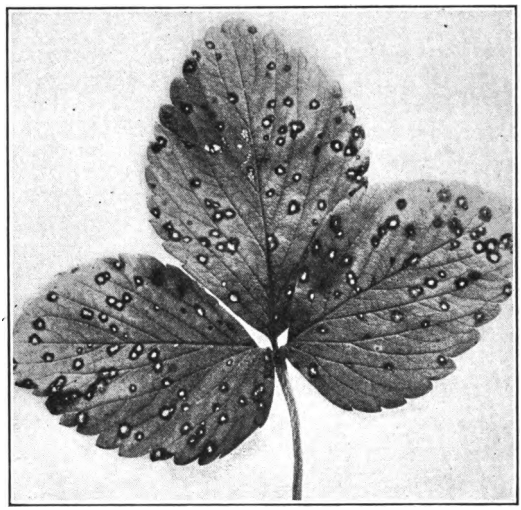

FIG. 123. — Strawberry leaf-spot.

It has been noted that conidial production ceases in the fall and that the winter is spent as mycelium in the old leaves. Toward winter the fungus makes preparations for other means of hibernation. Masses of mycelium appear on the surface of the leaf; these are sclerotial bodies which are capable of producing conidia in the spring following. A third means of over-wintering is by the perithecia; these arise in the fall, but remain immature until March or April, when mature asco-

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spores may be found therein. Unlike the ascospores of most ascomycetous fungi, they are not ejected from the perithecia nor are they blown to the new leaves. They germinate within the perithecia, even while yet in the asci, their germtubes emerging both through the ostiola and through breaks in the walls of the perithecia. At these points the germtubes cease growth and form conidia which are cut off and carried to the strawberry and cinquefoil. The first infections of the year occur most commonly about the time the fruit sets; later ones develop throughout the growing-season. The conidia germinate in the presence of moisture, and the tubes penetrate the plant more commonly through the cuticle, rarely through the stomata.

It has been shown that the mycelium does not descend to the roots or stems to hibernate. But, recapitulating, the winter is spent as follows: (1) as mycelium in the leaves; (2) as sclerotial bodies on the surfaces of the leaves; (3) as immature perithecia in the leaves.

Control.

In selecting varieties for planting, avoid the more susceptible sorts. Set in well-drained soil, and use only healthy plants. Remove diseased leaves before setting. Spray with bordeaux mixture 4–4–50 before the blossoms open to protect the plants against first infections. Repeat the application two or three times as the occasion demands. In ordinary cases, the first spraying is done just after the fruit is harvested. If there is an unusual prevalence of leaf-spot, renew the settings annually. Mowing and burning after harvest will often given a new set of leaves which go into the winter quite free from the fungus.

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Powdery-Mildew

Caused by *Sphærotheca Humuli* (Fries) Burr.

Strawberry powdery-mildew was first reported from England in 1854, at which time it was of considerable importance. In 1885 it again became noticeable in England. The next year it was found in America (New York State). In 1892 it was recorded from Massachusetts, but prior to 1896 the disease was rarely serious in the United States. Now the disease is common and widespread and is capable of doing serious damage.

Sometimes all varieties are affected; again certain ones are immune. Among the susceptible varieties may be noted: Paxton, British Queen, Black Prince, Jucunda, Mount Vernon, May King and Garrettson. On the other hand, the Noble and Royal Sovereign are said to be resistant.

Symptoms.

In the first stages the leaves are affected. They curl at the margin and thus expose their lower surfaces. Affected plants present an appearance as in need of water. Powdery, white growth of the mildew-pathogene appears on the lower surface. This is frosty and mealy in aspect. Berries are affected in all stages of development. They are rendered tasteless and

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worthless. Affected fruit dries up and neither colors nor matures. Its surface becomes covered with powdery-mildew, as described for the leaves. The fruiting-stalks are also affected.

Cause.

The causal fungus, *Sphærotheca Humuli*, grows superficially on the various organs of the strawberry plant. The powdery-mildew growth is composed of mycelium, conidiophores and conidia. At frequent intervals the mycelium sends haustoria into the epidermal cells for the purpose of obtaining food. As a result of this drain, the leaves curl and the plant suffers generally. The conidia produced on the leaves are blown to other plants, and infection may result. It is held by some that this same fungus attacks hops. Others believe that while the fungi on hops and strawberries are alike in form and structure, they will not pass back and forth, that is, the fungus on hops cannot infect the strawberry, and vice versa. This point needs further study. Later in the summer the pathogene develops perithecia. These are formed in the mycelial mats on the surface of the fruit, on the leaf-blades and leaf-petioles. These bodies carry the fungus through the winter. In the spring, ascospores are liberated and are carried to the growing plants, where infection results.

Control.

Little is known about the control of strawberry-mildew. The application of sulfur dust is effective on hops.

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Botrytis-Rot

Caused by *Botrytis* sp.

This disease is especially common and destructive in rainy seasons. It appears to have an extensive range over the country, occurring as far south as Louisiana and throughout the northern strawberry-growing sections. It was especially destructive over its entire range during the seasons of 1914, 1915 and 1916. It is reported as having caused a loss of millions of dollars in the States of Mississippi and Louisiana alone in 1914. Much of this loss resulted from the development of the disease during shipment, though it was prevalent and destructive in the field.

Symptoms.

All the above-ground parts are subject to the disease, but the fruit and fruit-pedicels usually suffer most. Small green fruits as well as mature and ripening berries may be affected. The lesions usually appear first on the fruit as small, brown, rotten spots. These rapidly involve the entire berry, from which the fruit-pedicels become affected. Leaves and leaf-petioles may show lesions, especially where they touch rotten fruits. The injury is a dry, brown rot. The fruit shrivels slightly, but retains its form. No exuding of juice is evident. The diseased tissue turns brown, but remains firm. The berry is finally coated with a dense growth of the spore stalks of the pathogene, giving it a grayish brown, moldy appearance.

Cause.

The fungus responsible for this disease is a species of Botrytis, possibly *Botrytis cinerea.* Another fungus *Rhizopus nigricans*(?) is also known to cause a rot of strawberry fruits which differs strikingly from the Botrytis-rot in that there is a rapid loss of juice from the fruit, giving it the name of "leak." This is accompanied by a collapse or flattening of the fruit. The Rhizopus rot appears more commonly on berries in transportation.

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The spores produced in great abundance from the spore stalks on diseased fruits are scattered through the berry patch or field, and many fruits become inoculated. When moisture is abundant and retained on the plants through the day, the spores readily germinate. Whether the germtubes can penetrate the uninjured surface of fruits is not certain, though this is probable. The mycelium spreads rapidly and profusely throughout the tissues of the fruit, between and through the cells, which are rapidly killed. The fungus feeds upon the substance of the invaded tissues and soon sends forth great numbers of brown stalks, branched at the tips and bearing thereon many grape-like bunches of oval thin-walled spores. These spores are readily scattered by the wind and cause new infections. How this fungus winters is not certain, though probably by means of sclerotia. Sclerotia are black tuber-like bodies of densely interwoven mycelium which are formed by all species of Botrytis, in dead host-débris. They pass the winter in a dormant condition, and germinate the following spring, producing tufts of spore stalks bearing spores like those already described. These spores initiate new infections.

Control.

Little definite information based upon experimental data or experience can be offered. Losses in shipment may be reduced by careful sorting out of all diseased or injured berries. Discarded fruit should not be allowed to accumulate about the packing house, but should be burned or buried daily. Field losses will be much reduced by growing the plants well apart in order to afford a maximum of light and air.

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Frost-Injury

Caused by the action of low temperatures

Spring frosts often do damage to strawberry plants. Those varieties in which the seeds are most exposed suffer most. Plants severely affected with leaf-spot have been observed to be more injured in the winter than those plants free from this disease. Mulching is practiced as a measure against winter-injury, while simply covering the plants with straw or blankets is practiced as a protection measure against spring frosts. (Fuller discussion of frost-injury given under Apple, page 35.)

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CHAPTER XVI

FUNGICIDES, THEIR PREPARATION AND APPLICATION

Most fungous diseases of fruits and fruit-trees are more or less profitably controlled by the application of external protective substances known collectively as fungicides. The essentials of a good fungicide are: it must have ability to protect the host from the attacks of the fungi; it must be non-injurious to the host; it must be adhesive, *i.e.* not readily washed off by rains; and must be relatively inexpensive and easy of application.

A fungicide usually acts to protect a host by destroying or inhibiting, through some toxic property, the growth of the fungous spores which fall on the exposed surfaces after the fungicide has been applied. Fungicides do not penetrate the host and kill the fungus after it has entered and established itself, hence the futility of applications after the host is infected. Fungicides prevent, they do not cure. In the case of a few fruit diseases, the fungicide acts as a disinfectant rather than as a protector, *i.e.* it destroys the spores of the pathogene which are already present when applied. The control of peach leaf-curl or of the surface-growing powdery-mildews are cases of disinfection rather than protection. Pathogenic fungi vary considerably in their sensitiveness to fungicidal poisons, both as to kinds and strengths, hence the necessity for using different kinds and strengths of fungicides for different fungous diseases. Sulfur fungicides, for example, so effective against apple-scab are of no value for the control of bitter-rot, against which copper fungicides must be used.

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That a fungicide must be non-injurious to the host is self-evident. Hosts, like pathogenes, vary in their sensitiveness to fungicides. Some, like the peach, are notably susceptible to injury from almost any toxic substance, while others, like the apple, will tolerate a wide range in kind and concentration of fungicides. Varieties and even individual trees exhibit great variation in their susceptibility to injury, so that only after extensive testing may one say with safety what kinds and strengths are to be applied to the different fruits. Moreover, the liability to injury depends on a number of variable conditions, such as the stage of seasonal development of the host, or its vitality, climatic conditions, especially temperature, moisture and the like. It is common knowledge among growers that in rainy seasons bordeaux mixture is apt to be very injurious to apples, while in dry seasons little or no injury follows its application. Foliage on apple-trees of low or impaired vitality is much more liable to lime-sulfur injury than that of healthy vigorous trees. Grapes of American origin are almost uniformly injured by the application of sulfur in any form while showing no evidence of injury from the application of copper fungicides. European grapes, on the other hand, are not injured by sulfur. Young, expanding leaves of the apple will tolerate a much more concentrated solution of lime-sulfur than will the fully expanded and mature leaves later in the season, strange as this may seem.

Adhesiveness, especially where the fungicide is to act in a protective way, is a very necessary requisite of a good fungicide. The spores of most pathogenic fungi are disseminated and infect the host during rains. Not only must the fungicide be present on the host in advance of the rains, but at least enough of it must remain, in spite of the washing of the rain, to destroy or inhibit the growth of the spores. Adhesiveness may be due to the gelatinous nature of the fungicide as in the case of bordeaux mixture, or to the finely divided character

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of the resulting particles as in lime-sulfur. Other substances having adhesive qualities are often added to assist in holding the fungicide in place on the host surface. Iron-sulfate and arsenate of lead act in this way when added to lime-sulfur.

The cost of the fungicide is often a limiting factor. A substance ideal as a fungicide in every other respect may cost more than is warranted by the profits realized from its application. Under average conditions, however, this is rarely the determining factor.

The ease with which a fungicide may be applied or the simplicity of the operations involved are of much importance in determining its general and effective use. The ease and rapidity with which dust fungicides may be applied in contrast to the slow and laborious application of liquid sprays will have much to do with the substitution of spraying by dusting.

Types of Fungicides

The fungicides used on fruits are of two general types, depending on the active principle in each: the copper fungicides and the sulfur fungicides. Although there are a number of kinds of each, bordeaux mixture, ammoniacal copper carbonate and water solutions of copper-sulfate are the copper forms usually used against fruit-diseases; while lime-sulfur, self-boiled lime-sulfur, soda-sulfur compounds and finely divided sulfur-dust are the usual forms of sulfur fungicides employed.

So far as fruits are concerned, it may be said that, in general, copper fungicides are always to be used on American grapes and are safe on sour cherries, gooseberries, currants, pears, quinces, strawberries and, in dry seasons, on apples. Sulfur fungicides alone may be used on peaches, plums and sweet cherries, and are also safe and effective on European grapes, apples, quinces, pears, cherries, currants and gooseberries.

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In the dormant condition, where disinfection is necessary, either copper or sulfur fungicides are safe and effective.

As regards the physical form, fungicides are of two general types, liquid and dry, or dust. Liquid fungicides have been used almost entirely since the introduction of bordeaux mixture about 1883. Several attempts to introduce copper fungicides in dust form have failed. Recently, with the development of sulfur fungicides, very successful and promising results have been obtained in the application of dry, finely divided sulfur.

Insecticides may usually be safely mixed with fungicides. Arsenate of lead is the insecticide now commonly employed. It may be used with bordeaux, lime-sulfur, and in powdered form with finely divided sulfur for dusting purposes. It may not be safely combined with soda-sulfur compounds.

Fungicides of all types are now generally manufactured, although they may all be home-prepared. The standard commercial brands are all about equally efficient and safe. They are in general more expensive than home-made preparations, but at the same time they are usually more convenient, especially where relatively small quantities are needed.

Bordeaux mixture was from 1885 to about 1910 the chief fungicide for fruit-diseases. With the introduction of lime-sulfur as a summer spray, bordeaux has taken second place. It is by no means, however, entirely replaced by lime-sulfur, and remains for American variety of grapes at least the only safe and effective fungicide, while for all other fruits except peaches, plums and apples (Fig. 126) it is just as safe and effective as any sulfur spray. The active principle in bordeaux is of course the copper. This is combined with the calcium in the form of gelatinous colloid membranes suspended in the water. The smaller the membranes, the better the mixture stands up, and the more effectively it covers sprayed surfaces. As the water evaporates from a sprayed surface, these minute

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membranes dry down and attach themselves like glue to the surface. When wetted by dew or rain, they swell but do not dissolve slowly. The action of the moisture and the air is to free a portion of the copper in the membrane which acts on the

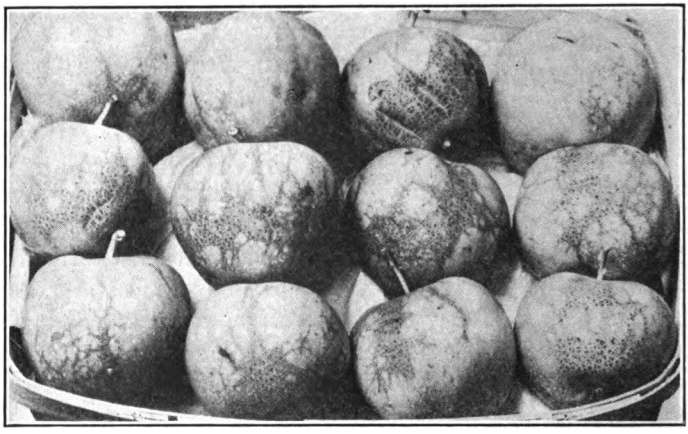

FIG. 124. — Bordeaux-injury on apples.

germinating spore to inhibit its growth or to kill it. Injury by bordeaux (Fig. 124) results when weather conditions favor excessive solution of the copper from the membranes.

FORMULÆ FOR PREPARATION

Bordeaux mixture.

The standard formula for use on fruit is usually a 5–5–50 or a 3–3–50 mixture, that is, 5 pounds quick-lime, 5 pounds copper-sulfate crystals, and 50 gallons of water; or if a weaker solution is desired, 3 pounds quick-lime and 3 pounds copper-sulfate to 50 gallons of water. In general, it is best to prepare stock solutions of the lime and of the copper-sulfate from which the bordeaux mixture may be prepared later as needed. To pre-

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pare stock solutions, proceed as follows: dissolve the required amount of copper-sulfate in water in the proportion of one pound to one gallon several hours before the solution is needed, suspending the copper-sulfate crystals in a sack near the top of the water. A solution of copper-sulfate is heavier than water. As soon, then, as the crystals begin to dissolve the copper solution will sink, bringing water again in contact with the crystals. In this way the crystals will dissolve much sooner than if placed in the bottom of the receptacle of water. In case large quantities of stock solution are needed, two pounds of copper-sulfate may be dissolved to one gallon of water.

Slake the required amount of lime in a tub or trough. Add the water slowly at first, so that the lime crumbles into a fine powder. If small quantities of lime are used, hot water is preferred. When completely slaked or entirely powdered, add more water. When the lime has slaked sufficiently, add water to bring the mixture to the proportion of one pound of lime to one gallon of water. These stock mixtures should not be allowed to dry out and should be kept covered.

To make the bordeaux, use five gallons of stock solution of copper-sulfate for every fifty gallons of bordeaux required. Pour this into the tank. Add water until the tank is about two-thirds full. From the stock lime mixture take five gallons of the thoroughly stirred lime-milk for each fifty gallons of the mixture required. Dilute this a little by adding water, and strain into the tank. Stir the mixture and add water to make the required multiple of fifty gallons. Experiment stations often recommend the dilution of the copper-sulfate solution and the lime mixture each to one half the required amount before pouring together. This is not necessary, and is often impracticable for commercial work. It is preferable to dilute the copper-sulfate solution. One should never pour together the strong stock mixtures and dilute afterwards. Bordeaux mixture of other strengths as recommended is made in the

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same way, except that the amounts of copper-sulfate and lime are varied according to the requirements.

Copper-sulfate.

This salt alone may be used only in very dilute solutions on foliage and fruit, and as a summer spray has but few uses. As a disinfectant for dormant peach trees it may be used as strong as necessary and is very effective against peach leaf-curl.

Copper-sulfate, 1 lb.; water, 15–25 gal. Dissolve the copper-sulfate in the water. It is then ready for use. One pound in twenty gallons of water has been found effective against the above named disease.

Lime-sulfur solution.

The fungicidal value of lime-sulfur was discovered in America about 1880 when California peach-growers using it on dormant trees for the San José scale found that it controlled the leaf-curl. As a matter of interest it may be noted that several years earlier a boiled solution of lime and sulfur much diluted was in use by European gardeners as a fungicide on growing plants. Its use was, however, not general and its possibilities were disregarded or overlooked in the general belief that the newly discovered bordeaux mixture was of universal application. The re-discovery of lime-sulfur as a summer spray about 1906 by Cordley of Oregon marks the beginning of a new epoch in the history of fungicides.

The fungicidal properties of lime-sulfur reside in the sulfur or, more accurately perhaps, in the sulfuric acid which is eventually formed by the oxidation of the sulfur in the presence of water. The special virtue of the lime-sulfur solution (polysulfides of calcium) lies in the fact that after evaporation of the water they are gradually oxidized, leaving pure sulfur in exceedingly finely divided particles on the sprayed surface. Injury by lime-sulfur usually occurs as the solution dries, and especially where the trees have been drenched. Large quantities of the solution are left along the curved edges of the leaves, where as

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evaporation goes on concentration and burning by the caustic polysulfides results. In the case of grapes, however, and sometimes in the case of burning of peach and apple foliage, the injury is due to the sulfuric acid formed later from the sulfur. If the lime-sulfur is properly diluted and properly applied, serious injury seldom results except on grapes and on some varieties of peaches. What has sometimes been taken for lime-sulfur injury is doubtless injury from the arsenicals used with it.

If commercial concentrated solutions are to be used, simply dilute as directed for the particular disease or diseases to be combated. Concentrated solutions may be prepared as follows:

Use only fresh, lump-lime free from dust, grit, air-slaked material and magnesium oxid. The lime should not contain less than 90 per cent of pure calcium-oxid. A high grade of sulfur flour, flowers of sulfur, or finely ground brimstone will do equally well. The best formula appears to be:

Lime 90 per cent pure,	40 lb.
Sulfur	80 lb.
Water	50 gal.

Moisten the sulfur thoroughly, working it into a smooth, even fluid-paste. Slake the lime in 10 gallons of hot water, adding the lime slowly to avoid boiling over. Pour in the sulfur paste gradually during the slaking, and stir constantly. When all the lime is slaked and all the sulfur thoroughly mixed in, add water to make fifty gallons and boil vigorously for one hour. The cooking may be done in a kettle over a fire or in a barrel by means of live steam. Allow the solution to settle, decant off the clear dark concentrate, and store in barrels. Keep where it will not freeze. Home-made concentrate will usually show a lower Baumé test than the commercial concentrate. In either case the dilution should be made on the basis of the hydrometer reading.

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Self-boiled lime-sulfur.

This fungicide is not a boiled solution, as might be inferred from the name. It is in reality a mixture of lime and sulfur resulting from the violent action of slaking lime in the presence of finely divided sulfur. It was devised by Scott about 1907 for spraying peaches to protect them from the brown-rot. When properly made it is the safest and most efficient fungicide for the summer spraying of peaches.

Prepare self-boiled lime-sulfur as follows: place ten pounds of sulfur and ten pounds of stone-lime in a barrel. Add cold water slowly to slake the lime, keeping the mass wet but not submerged. Stir occasionally. Part of the large lumps of lime may be kept out at first and added after slaking has progressed to some extent, thus prolonging the slaking and heating. When slaked, dilute at once to fifty gallons, and apply as in the case of bordeaux.

Soda-sulfur compounds of various types and under various names are on the market. They are probably as effective as the lime-sulfur compounds, but are more likely to cause injury to the foliage. They must generally be used in greater dilution and may not be combined with arsenate of lead. The so-called "soluble-sulfur" and "sulfocide" are of this type.

Sulfur-dust.

Sulfur for dusting purposes must be especially fine. Finely ground sulfur-flour is preferable. Powdered arsenate of lead in the proportion of ten pounds to ninety pounds of sulfur should generally be used, not only because of the insecticidal value of the arsenate of lead, but also because of its effect on the physical condition of the sulfur. By combining powdered arsenate of lead and sulfur-flour, the resulting dust is fluffy and consequently better distribution and better adhesive qualities are obtained. The dilution of the sulfur with finely powdered lime is not to be recommended because the lime appears to have a detrimental effect on the fungicidal action of the sulfur.

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Finely powdered gypsum or other inert carriers may be added, but their use is of doubtful value.

Iron-sulfate.

This salt has of itself little value as a fungicide, but added to lime-sulfur solution forms an iron-sulfide. It reduces the burning qualities and increases the adhesiveness of lime-sulfur. It appears that iron-sulfide is quite as effective as lime-sulfur. It is made by adding $2\frac{1}{2}$ to 3 pounds of granulated iron-sulfate to each 50 gallons of diluted lime-sulfur solution.

Application of Fungicides

Fungicides, to be effective, must be properly applied. There are two important factors never to be neglected. They may be designated as timeliness and thoroughness.

Having determined on the proper kind of fungicide and the correct strength to be used in the case in hand, the question of the time of application comes up for consideration. Timeliness in making the application is the key to success in the control of diseases with fungicides. The stage of the development of the host must be the primary consideration; the tree, not the calendar, must be the guide. For example, the time to spray apple-trees to protect from scab is determined by the stage of development of the blossoms or fruit; first application, just before the blossoms open when the central blossom shows color and after the individual blossom-buds in the cluster have separated (Fig. 124). Second application, just after the petals fall (when two-thirds off) (Fig. 125), and so on. One must watch the trees closely and act promptly when they are just right for application. Seasons differ, varieties differ, and these facts must be taken into consideration. Next to the stage of the development of the host, as determining the time to spray, comes the weather. Spray just before rains, not after them. Remember that the fungus usually reaches

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the leaf or the fruit and produces infection during the rain, not before nor after. Get the fungicide on ahead of the rain and thus ahead of the fungus. Watch the weather maps and the developing blossoms. It will be more profitable in some cases to spray a little before the host is in just the right condition in order to get in ahead of a rain-period. Long, rainy, cloudy periods are the dangerous ones. Heavy showers followed by rapid clearing seldom afford conditions favorable to serious infection by orchard fungi.

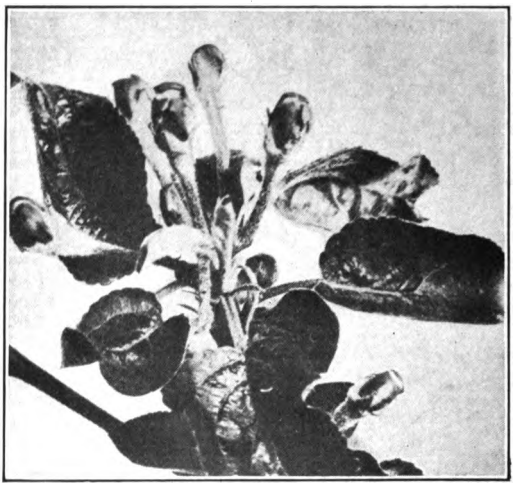

FIG. 125. — Apple-blossoms in proper stage for the first application of a fungicide.

The period for effective applications of fungicides, in practically all cases, is a brief one; at most a few days, more often only a day or two. This means that equipment, labor and materials necessary to cover the trees in a short time must be provided. The continuous running of one sprayer in a large orchard throughout the season is largely a loss of time and money. Fungicides to be effective must be applied at just the right time.

Thoroughness is second only to timeliness as a factor in determining the success of spraying or dusting operations. Since fungicides are applied to protect, every part of the susceptible surface must be covered. In spraying this cannot be

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done rapidly. Dusting can be done in much less time. Spraying will be much more thorough if done against, rather than with, the wind. Use a nozzle set at an angle of 60° on a ten- to fourteen-foot pole with a pressure of 175–200 pounds behind it. The spray should be fine and the nozzle should be

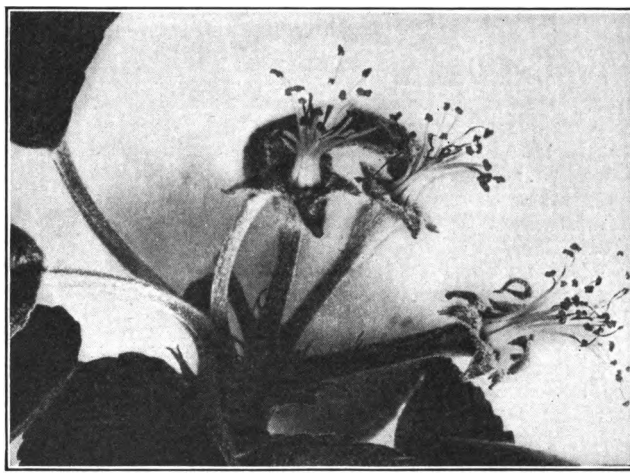

FIG. 126. — Apple-blossoms in proper stage for the second application of a fungicide.

moved along carefully and intelligently over every limb and branch. Where trees are fifteen feet or more in height, spraying should be done from a tower, and where the trees are very large, a man on the ground with a trailer will be necessary to cover the low-hanging limbs and lower inner branches. In spraying against the wind hold the nozzle at such a distance from the limb to be sprayed that the spray will come just where the wind breaks the force of the spray. Where spraying is done with the wind a second application after the wind changes is necessary. This is usually too late to be effective and requires double the time and material. Dusting is done with

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the wind, but here the light particles floating slowly through the branches settle and coat every part. Dusting is to be done at the same times that spraying should be done. However, since it can be done much more rapidly, the grower may often be able to make a timely application of dust which would not be possible with spray.

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APPENDIX

SOME BOOKS THAT CONTAIN INFORMATION ON FRUIT DISEASES

THE books listed below will be found helpful to any one who is especially interested in fruit diseases.

Duggar, B. M. Fungous Diseases of Plants, pp. 1–508. (Ginn and Company.) 1909.

Freeman, E. M. Minnesota Plant Diseases, pp. 1–432. 1905.

Stevens, F. L., and Hall, J. G. Diseases of Economic Plants, pp. 1–513. (The Macmillan Company; New York.) 1910.

Cooke, M. C. Fungoid Pests of Cultivated Plants, pp. 1–278. (Spottiswoode and Company; London.) 1906.

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McAlpine, D. Fungus Diseases of Stone-Fruit Trees in Australia and their Treatment, pp. 1–165. (Melbourne, Victoria; Department of Agriculture.) 1902.

Scribner, F. L. Fungus Diseases of the Grape and other Plants and their Treatment, pp. 1–134. 1890.

Lodeman, E. G. The Spraying of Plants, pp. 1–399. (The Macmillan Company; New York.) 1896.

Peets, Elbert. Practical Tree Repair, pp. 1–265. (McBride, Nast and Company; New York.) 1913.

SOME JOURNALS THAT CONTAIN INFORMATION ON FRUIT DISEASES

The Gardeners' Chronicle.

Phytopathology.

Zeitschrift für Pflanzenkrankheiten.

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GLOSSARY

Acervulus (acervuli). Open, saucer-shaped, asexual fruiting body. See pycnidium.

Æciospore (æciospores). Spore borne in an æcium.

Æcium (æcia). Type of fruit-body in rusts.

Annulus (annuli). A ring-like portion of a mushroom veil, remaining attached to the stem after its rupture and after the expansion of the cap, or pileus.

Apothecium (apothecia). Open, cup-shaped, sexual fruit-body. See perithecium.

Appressorium (appressoria). Hold-fast, or organ of attachment developed by certain fungi.

Ascospore (ascospores). Spore borne in an ascus. See ascus.

Ascus (asci). Sexual spore-containing sac, usually developed within an apothecium or a perithecium.

Bacterium (bacteria). A simple plant of low order lacking chlorophyl, or leaf-green. It has no mycelium and each plant is but a single cell. Reproduction by fission. Spores sometimes produced. See fungus.

Calyx (calyces). Outer series of leaves of a flower, usually green.

Cambium. The growing and formative tissue of a stem or root, found between the wood and bark.

Canker. A definite dead area in the bark of trees or shrubs.

Cap. See pileus.

Chlamydospore. Thick-walled asexual spore which usually is capable of resting.

Chlorophyl. Green coloring-matter in leaves.

Cilium (cilia). Thread-like process on bacteria or motile spores which aids in motility.

Conidiophore. A spore-bearing stalk.

Conidium (conidia). Asexual spore.

Cortex. Outer bark.

Crenulate. Finely notched.

Cuticle. A continuous waxy film covering the surface of plants.

Enphytotic. Said of a plant disease which is habitually and permanently prevalent in a locality, although not unusually destructive. See epiphytotic.

Enzyme. A chemical compound capable of assisting or hastening chemical transformation, but without itself entering into the final product.

Epiphytotic. Said of a plant disease which suddenly appears in a locality in an unusually destructive manner. See enphytotic.

Fungus (fungi). A simple plant of low order lacking chlorophyl, or leaf-green. It has mycelium which corresponds to roots. Reproduction by means of spores. See bacterium.

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Fruit-body. A spore-bearing structure.
Fusiform. Spindle-shaped.
Germtube. A tube developed from a spore on germination. It usually gives rise directly to mycelium.
Gill. Plate or lamella of a mushroom, found on the lower side of the cap or pileus.
Haustorium (haustoria). Special mycelial branch sent into the cell serving as an organ for obtaining food.
Heterœcious. Said of fungi (usually rusts) requiring more than one kind of host-plant on which to complete the life-history.
Hypertrophy. Abnormal enlargement of a part or the whole of a plant. Specifically, the abnormal enlargement of cells.
Hypha (hyphæ). A vegetative or mycelial thread of a fungus. See mycelium.
Infect. To cause disease.
Infection. Act and condition of being infected. See infect.
Infection-court. Place where infection is initiated.
Inoculum. The transferable portion of a pathogene which may bring about infection.
Lamella (lamellæ). Gills of a mushroom. See gill.
Lenticel. A specially developed loose cellular structure in the cork of a plant through which an exchange of gases between the interior of the plant and the air may take place.
Lesion. Any definitely diseased region.
Middle-lamella. The primary membrane between any two cells of higher plants.
Morphology. The science which treats of the form, size and color of an organism.
Mycelium. The vegetative system of a fungus made up of hyphæ or threads. See hypha.
Oospore. Special type of sexual spore.
Parasite. An organism which lives in or on another living organism during a portion or all of its life for the purpose of obtaining food and habitation. The latter organism is referred to as a host. A parasite is usually smaller than its host.
Pathogene. A disease-producing factor, usually an organism.
Pedicel. A fruit-bearing or flower-bearing stalk.
Peduncle. A general stalk bearing a group of flowers or fruits. May bear but one flower or fruit.
Perennial. Lasting more than two years.
Perithecium (perithecia). Closed globose or flask-shaped, sexual fruit-body containing asci. Finally a small opening or pore may appear at the top. See apothecium.
Petal. The inner row of flower leaves, usually white or colored other than green.

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Pileus. Cap of a mushroom.

Promycelium. A special type of germtube of the rust and smut fungi. It forms a basidium with basidiospores or sporidia.

Protoplasm. Living substance of an organism.

Pycnidium (pycnidia). Closed, globose or flask-shaped, asexual spore-containing body. See acervulus.

Pycnosclerotium (pycnosclerotia). Sclerotial body resembling a pycnidium which may become a perithecium.

Pycnospore. An asexual spore — a conidium — borne in a pycnidium.

Rhizomorph. A root-like, branched strand of fungus hyphæ.

Saprophyte. An organism living on dead material.

Sap-wood. Living wood next to the bark which contains much of the sap.

Sclerotium (sclerotia). A tuber-like vegetative structure of certain fungi consisting of a densely woven mass of hyphæ which may rest for a long period.

Seta (setæ). A bristle, or bristle-like body.

Sporangium (sporangia). Asexual spore-containing sac. Not inclosed.

Spore. A cell capable of reproducing a plant like the parent. A structure belonging to the fungi and related plants. Corresponds in function to a seed.

Sporidium (sporidia). Diminutive of spore. Usually applied to spores of the rust or smut fungi borne on the promycelium.

Stoma (stomata). A minute opening in the epidermis of stems, leaves and fruits which admits free exchange of gases between the interior and exterior.

Stroma (stromata). A fruiting, or spore-bearing, cushion composed of mycelium entirely, or of mycelium and host-tissue.

Swarm-spore. Motile spore.

Teliospore. Chlamydospore of a rust fungus which usually rests. Produces a promycelium on germination.

Telium (telia). Sorus in which teliospores are borne.

Uredinium (uredinia). Sorus in which uredospores are borne.

Uredospore. Spore of a rust fungus which perpetuates it throughout the summer. Produces a germtube on germination.

Veil. A special membranous envelope in mushrooms, which at first covers the gills. Later remnants of it may be seen on the stalk or stipe.

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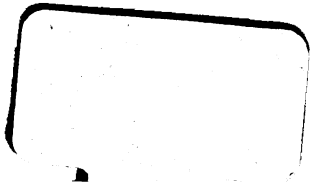

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